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FRACTURES IN THE NECK OF THE FEMUR IN CHILDREN WITH PARTICULAR REFERENCE TO ASEPTIC NECROSIS*

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Fracture of the femoral neck in children, though a rare injury, frequently results in permanent disability. The characteristic site is at the cervicotrochanteric junction, and the most common cause is severe trauma received by falling from high elevations. Satisfactory reduction has been more difficult to secure and retain in children than in adults by the same commonly employed methods of treatment. Finally the incidence of aseptic necrosis is probably higher than in fractures of the neck of the femur in adults.

In this study of twelve such fractures in ten children the authors conclude that reduction with application of the Whitman abduction plaster is inadequate; nailing is undesirable; and traction alone does not correct the deformity. Traction and abduction combined not only reduce the fracture accurately, but maintain it satisfactorily. The location of the fracture in the cervicotrochanteric region is a favorable site for circulatory damage and aseptic necrosis.

A summary of this group, with regard to location of the fracture, shows eight of the cervicotrochanteric type and four of the mid-cervical type. Each of the latter four cases had some complicating factor relating to the injury.

Of the several methods employed for reduction and maintenance, manipulation followed by limited or full abduction in plaster was used in four cases of cervicotrochanteric fractures with poor results, and in two mid-cervical fractures with good results. The Hoke-type plaster with well-leg countertraction was used late in one case and was not successful. Had the adductors been tenotomized and the leg placed in wide abduction, the method would probably have succeeded, even though applied twenty

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FIG. 1-A

Case 1. Roentgenogram taken February 8, 1926, twelve months after cervicotrochanteric fracture. There has been increasing lameness for three months. Aseptic necrosis is well marked.

FIG. 1-B

Case 1. Roentgenogram taken March 18, 1927.

days after injury (Case 3). Abduction with traction was applied primarily in four instances and gave excellent results. In another case abduction with traction was applied with satisfactory results two weeks following injury, because of recurrence of the deformity (Case 4). A total of five fractures were reduced anatomically and held until union was complete by the method of abduction and traction. There was some variation in the method for securing abduction and traction in this group of four cases and five hips, but equally good results were obtained. In Case 1, Buck's extension with considerable weight, and elevation of the foot of the bed for six weeks, was used. In Case 4 the deformity recurred with the leg in plaster in fair abduction. Two weeks later a new plaster was applied which included both legs with wider abduction, skeletal traction through the lower end of the femur, and direct pull with weight for five or six weeks. Excellent position was secured, though aseptic necrosis later developed. In Case 10, a Jones splint was applied with skeletal fixation on the injured side. A Roger Anderson splint should be as effective. In Case 9, which was a bilateral one, fixed traction in wide abduction was used. Reduction was secured by table traction and abduction. Adductor muscles were then very tight and were tenotomized. A Kirschner wire was placed through the lower end of each femur and double spicas applied. It is probable that the fixed traction in abduction without adductor tenotomy would have been adequate, but with the high incidence

FIG. 1-C

Case 1. Roentgenogram taken fifteen years after injury. Patient has a slight limp but free motion and no pain.

FIG. 2-B

Case 3. Roentgenogram taken January 30, 1934, shows open reduction and pin fixation.

FIG. 2-A

Case 3. Roentgenogram taken December 30, 1933, three weeks after injury, shows Hoke traction applied. Note distraction at the joint.

FIG. 2-D

Case 3. Roentgenogram taken October 25, 1935. Patient has instability and a painful limp.

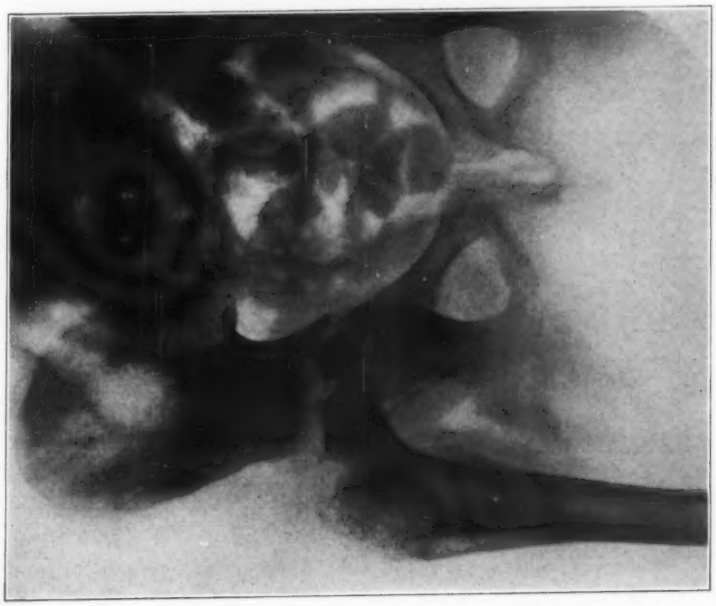

FIG. 2-C

Case 3. Roentgenogram taken April 4, 1934, shows aseptic necrosis.

of recurrence into deformity we would employ the same technique in another bilateral case. Convalescence was not appreciably affected by weakness in adduction.

Of the three patients who had open reduction, one, with fixation by a Smith-Petersen nail, had a successful result; in one, pin fixation was a failure because of aseptic necrosis; and one, with no fixation by internal device, had a very poor result.

In this group of twelve fractures, eleven were treated and the end result, regardless of complications, was good in seven, fair in two, and poor in two. Two in the group recorded "good" have been followed less than one year and aseptic necrosis may yet occur and alter the results. There was only one definite non-union and this was complicated by aseptic necrosis.

It is apparent that two major problems are encountered in treating these cases. It is believed that the first, relating to reduction and retention in the cervicotrochanteric type, has been solved by application of traction with abduction. Successful reduction can be accomplished in combination with plaster to permit early discharge from the hospital. The mid-cervical fractures may be treated by abduction and internal rotation, or, if necessary, by nail or pin fixation, though consideration must be given to premature closure of the epiphysis in the latter method. In patients with a deformity which has existed longer than four or five weeks, the fracture should be permitted to unite, and later a corrective osteotomy may be performed.

FIG. 3-A

Case 4. Roentgenogram taken May 24, 1936, shows typical cervicotrochanteric type of fracture, which was treated in plaster for two weeks, and then by skeletal traction in abduction.

FIG. 3-C

Case 4. Roentgenogram taken October 9, 1936. Patient has good union, slight limp, and no pain.

FIG. 3-B

Case 4. Roentgenogram taken July 23, 1936. Note increased density in the neck.

TABLE I

RÉSUMÉ OF TEN PATIENTS WITH TWELVE FRACTURES OF THE FEMUR

Case	Sex	Age	Type of Fracture	Treatment	Reduction	Secondary Plan	Reduction	Union	Coxa Vara	Aseptic Necrosis	Result
1*	F	9	Cervicotrochanteric, right	Reduction, traction in abduction. Buck's extension with foot of bed elevated for six weeks	Yes			Yes	No	Yes	Fair, 15 years
2	F	11	Cervicotrochanteric, right	Reduction and plaster	No	Open reduction after six weeks	No	No	Yes	Yes	Poor, 10 years
3*	F	5	Cervicotrochanteric, right	Reduction and plaster	No	Open reduction after four weeks	Yes	Yes	Yes	Yes	Poor, 7 years
4*	M	11	Cervicotrochanteric, left	Reduction and plaster	No	Reduction, traction in abduction	Yes	Yes	No	Yes	Fair, 4 years
5	F	8	Cervicotrochanteric, right	Reduction and plaster	No	After four days second reduction and plaster	Yes	Yes	30 degrees	No	Good, 2 years
6†	F	14	Mid-cervical, bilateral	Reduction and plaster for both hips	Yes			Yes	No	No	Good, both hips, 18 months
7	M	15	Mid-cervical, left	None	Poor	Fused hip	Crutches	Yes	Yes	No	Good for fused hip, 1 year
8	M	11	Mid-cervical, right	Smith-Petersen nail	Yes			Yes	No	No	Good, 16 months
9*	M	8	Cervicotrochanteric, bilateral	Reduction, traction in abduction, tenotomy of tight adductors, skeletal fixed traction for eight weeks	Yes			Yes	No	No	Good, bilateral, 18 months
10*	M	8	Cervicotrochanteric, left	Reduction, traction in abduction	Yes			Yes	No	No	Good, 13 months

* Cases illustrated in the text.

† A spastic child wearing thigh-length braces fell to the floor and fractured one hip. One year later she again fell and fractured the other hip.

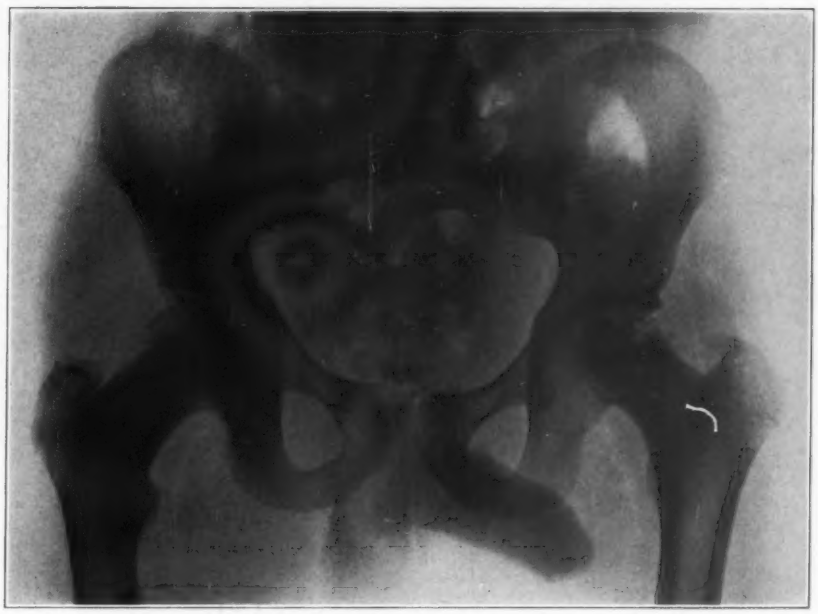

FIG. 3-D

Case 4. Roentgenogram taken December 29, 1937, four months after weight-bearing. Patient has slight limp, and limitation of motion, but no pain. Localized aseptic necrosis is present.

FIG. 3-E

Case 4. Roentgenogram taken March 5, 1940, four years after injury, shows no clinical change.

ASEPTIC NECROSIS

The second problem is aseptic necrosis. This may occur regardless of treatment, but results may be favorably altered by avoiding vigorous

FIG. 4-A

Case 9. Roentgenogram taken September 2, 1939, shows bilateral fracture. The patient fell twenty-five feet from a tree.

FIG. 4-B

Case 9. Roentgenogram taken November 16, 1939, after treatment by adductor tenotomy, abduction in plaster with skeletal fixation by wires at the lower end of the femora.

FIG. 4-C

Case 9. Roentgenogram taken February 22, 1940. Function was good for this date.

Note: Eighteen months after injury there was no evidence of aseptic necrosis, and function was good.

manipulation in primary reduction and by a long period of freedom from weight-bearing.

Because of the high incidence (33.3 per cent.) of this complication in the authors' series, several roentgenograms are reproduced which may be interpreted in relation to the circulatory mechanism in the femoral head of children.

Wolcott has shown, by injections of opaque materials, a pattern of anastomosis in the hips of children up to ten or thirteen years of age, which differs from that found regularly in 80 per cent. of adult hips.

In adults he finds that the capsular vessels enter, in two groups of three or four branches each, at the posterosuperior and postero-inferior juncture of the cervicotrochanteric region. They may traverse the loose capsular tissue to enter obliquely at the base of the head, and anastomose with the branches from the nutrient and the ligamentum teres vessels. In 20 per cent. of adult hips, the ligamentum teres vessel is absent, and in all children it is not patent beyond the fovea until the nucleus for the head is well formed at eleven to thirteen years of age, after which free anastomosis occurs.

On the basis of Wolcott's findings, the bone reaction in Case 3, aged five, may be interpreted as follows: The superior capsular vessels were torn with the original injury. The nutrient vessels, already injured, were probably obliterated by introduction of the pin. The ligamentum teres vessel at the age of five had not penetrated the head. The inferior cap-

FIG. 5-A

Case 10. Roentgenogram taken December 7, 1939, shows a typical fracture in a child of eight years, caused by a fall of two stories.

FIG. 5-B

Case 10. Roentgenogram taken December 20, 1939, shows gradual reduction by skeletal fixation of Jones splint on the fractured femur.

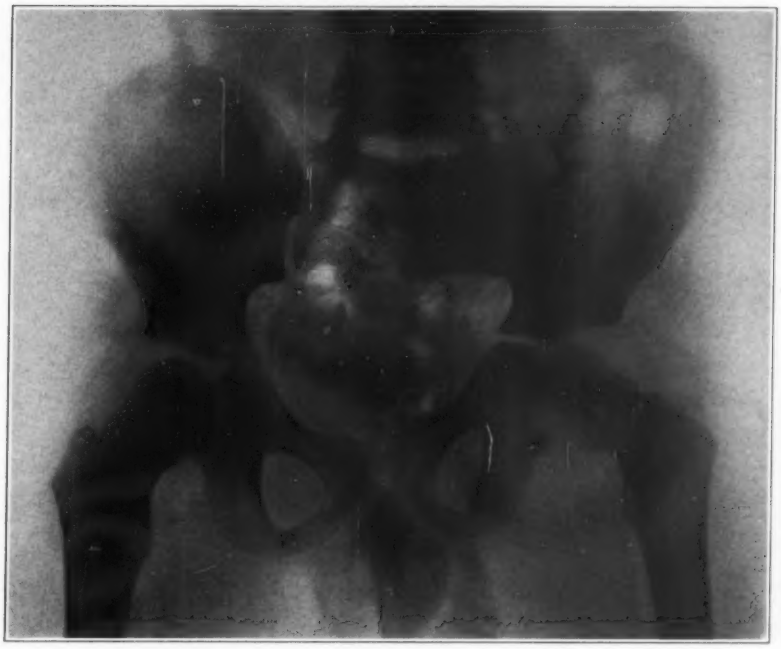

FIG. 5-C

Case 10. Roentgenogram taken February 15, 1940, shows firm union. Note: Thirteen months after injury function was good and there was no evidence of aseptic necrosis.

sular vessels, though damaged, were adequate for early union of the fracture but were not sufficient to support the neck and head (Figs. 2-A, 2-B, 2-C, and 2-D).

CASE 1. Twelve months after the fracture in a girl, aged nine years, there was extensive necrosis in the neck and marblelike density in the head (Fig. 1-A). It was apparent that the ligamentum teres vessels had not been functioning and that complete disintegration could be anticipated. This is shown in Figure 1-B taken thirteen months later. The neck was revascularized, probably through nutrient arteries (Fig. 1-C).

CASE 4. This case of a boy aged eleven may be interpreted with greatest accuracy. The several illustrations, Figures 3-A, 3-B, 3-C, 3-D, and 3-E portray healing reactions with indications of an early loss of the superior capsular vessels. Note the increased density in the upper two thirds of the neck, apparent for several months during the early period of convalescence. The appearance of necrosis in the head may be explained by assuming that at the age of eleven the ligamentum teres vessels had not entered the head and completed its anatomical connections. Had it been present before the accident, the upper quadrant of the head would have survived as did the damaged area of the neck.

Whitman[10], in 1893, called attention to these fractures in children, and during the next few years reported ten cases. Most of these were seen late, and because of coxa vara deformity. He suggested that such cases, if seen early, should be treated by wide abduction and, if separation were present, traction.

Taylor, in 1917, reported seven cases from the Hospital for Ruptured and Crippled. He observed that fractures in older children were similar

to those seen in adults. In the younger children the fractures occurred in the base of the neck, and there was a marked tendency to coxa vara. He advised abduction and plaster when the patient was seen early and had deformity.

Bland-Sutton, in 1918, reported two instances of fracture and called attention to the rarity of this lesion. The next year Greig reported six cases, but did not specify the particular treatment employed. One patient recovered with coxa vara deformity.

Whitman[12], in 1900, again discussed this subject in connection with slipped epiphyses in children. Most of the thirty-one cases were seen late and had considerable deformity. Several others[4, 7, 9] during this early period reported a few cases, usually seen late. They made no mention of changes which would suggest aseptic necrosis.

In 1928 and 1929, Colonna reported twelve cases, eleven of which were cervicotrochanteric in type. Results were good in seven and poor in five. The Whitman abduction plan of treatment was followed in most of the cases.

Mitchell, in 1936, reported nine cases. Only three were treated early, with two good results. The remaining six were seen late in deformity. Mitchell recognized the Hoke-traction plan as the treatment of choice.

John Wilson, in 1940, reported ten cases, emphasizing the difficulties in reduction, and the frequency of aseptic necrosis. He suggested that better methods should be devised for treating this serious injury in children.

Reports in the literature, therefore, should substantiate the conclusion that cervicotrochanteric fractures in children are not satisfactorily reduced and maintained in reduction by the method which has usually been employed in the treatment of fractures in adults.

SUMMARY

Fractures of the hip in children are of infrequent occurrence. The type of fracture is cervicotrochanteric in 75 per cent. of the cases.

Treatment by the methods usually applied in adult cases has not been satisfactory.

After reduction, fixation is best secured by abduction, plaster, and traction. Skeletal fixation in plaster may be substituted for traction.

Adductor tenotomy may be necessary in delayed reduction.

Cases seen late in coxa vara position (after four weeks) should be permitted to unite and then to have the deformity corrected by osteotomy; otherwise aseptic necrosis may supervene.

Aseptic necrosis is a frequent complication. To lessen the damage from it, a long period without weight-bearing should be advised.

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TREATMENT OF TENDONS IN COMPOUND INJURIES OF THE HAND

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TREATMENT OF WOUNDS

Severance of a tendon is an indication to hurry the patient to a hospital where good surgical facilities and surgical skill are available. Without these, suture of a tendon should not be attempted, as it does more harm than good. The fate of a tendon is dependent on the proper treatment of the wound as a whole, because a tendon cannot live long either when exposed or when bathed in infection. Therefore, when tendons are severed, the proper treatment of the wound in general is essential in order to provide an environment of clean primary healing.

In a compound injury of a hand, the outcome is determined by the selection of the primary surgical procedure. Thus, the patient may be spared long illness and extensive crippling by preventing sloughing tendons, infected joints, and osteomyelitis. This can be accomplished by observing the following three main principles: (1) early operation before the germs introduced have become numerous, (2) thorough débridement, and (3) covering over of all vulnerable tissues.

Time Factor

It is preferable to suture tendons within six hours after the accident,—that is, before the bacteria in the soiled wound have greatly multiplied. A tendon should never be sutured after twenty-four hours. Very bad infections frequently result from breaking this rule. In many cases the arbitrary six-hour limit, based on hospital statistics, is unnecessarily short because there are various determining factors other than time alone. If such factors are favorable and a smear from the wound does not show cocci or an excess of pus cells, tendon suturing up to twelve or even twenty-four hours is possible. Judgment in this should be based on the degree of trauma, amount of bacteria, virulence of bacteria, resistance of patient, and the method of wound handling and after-treatment. Infection is less apt to occur in clean cut wounds than in crushing or explosive ones, or those with dirt ground in, and less in wounds incurred indoors with clean tools than those received outdoors. The infections from a human bite or the bite of an animal incurred in a slaughter house are especially prone to be virulent. In a healthy, resistant individual, infection is less likely to occur, mildly contaminated wounds often healing *per primam*. Thus, by considering all factors, including management, primary suture of tendons can be successfully done after six hours, in a gradually decreasing number of cases, up to, but never after, twenty-four hours.

Débridement

Washing a wound with a large amount of normal salt solution does not constitute débridement, as it does not remove from the wound the surface tissues which have been traumatized. Unless careful and thorough excision is done, tendon suturing is frequently followed by severe infection, because the wound must rid itself of a layer of traumatized, infected tissue by necrosis and sloughing.

After the area surrounding the wound has been shaved and cleansed with soap and water, the limb is wrapped in a towel and wound bloodless from fingers to above the elbow with a rubber Esmarch bandage. One and one-half inches above this, where its pressure will not be influenced by removal of the Esmarch, the cuff of a blood-pressure apparatus is applied and pumped to 300 millimeters of mercury, the tubes are clamped and the Esmarch removed. An Esmarch tourniquet may cause paralysis, but a blood-pressure band, so used, will not. After local or block anaesthesia (never adrenalin in the digits), the surrounding area is painted with equal parts of tincture of iodine and alcohol, and the wound is iodized thoroughly.

The wound is débrided—that is, converted into a non-traumatized, aseptic wound—by excising its complete surface while sparing essential structures, such as nerves, blood vessels, and tendons. With sharp, flat, curved, double-pointed scissors, the skin edges are cut away and the whole surface of the wound is systematically excised to a depth of one or two millimeters, or more if the tissue is too badly traumatized. The infected bone ends are chiselled off thinly, the infected ends of tendons and nerves are shaved off, and all the iodine-stained tissue is removed, so that there remains a surgically aseptic, chemical-free wound of viable tissue.

Covering of Vulnerable Tissues

Tendons, nerves, joints, and bones cannot live exposed to the outside world and when infected cause sloughing, months of dressings, and final crippling. Before germs have multiplied all these structures should be covered by plastic skin flaps from the immediate neighborhood. The denuded areas from which the skin flaps are swung should be covered immediately by Thiersch skin grafts.

After the skin flaps are fashioned, the tourniquet is removed, and hemorrhage is checked by steady pressure for a few minutes, and a minimum of No. 000 catgut ties, or even better No. 36 or No. 38 stainless-steel wire which is hairlike and does not interfere with healing.

Postoperative immobility is strictly maintained for at least a week by a half circumference of plaster of Paris. The limb is kept elevated until the tendency to swell is over. Following the operation, mild gauze pressure is applied for a few hours or until the next day to prevent hematoma. The wound is then uncovered and allowed to dry, and is protected only by a wire cage covered by a towel or gauze. Even a dry gauze dressing over a wound becomes moist in twenty-four hours and encourages

infection to enter along the stitches. Dryness prevents this. Antitetanic serum should not be forgotten.

REPAIR OF TENDONS

Indications for Tendon Suture

In disability cases, coming later for reconstruction of solidly congealed and contracted hands, much of the destruction can be ascribed to the very bad infection that resulted from the additional trauma of surgery. Tendons in these hands were often sutured after twenty-four hours following the accident when bacteria were so numerous that they were spread and planted extensively through the hand and wrist by the surgery.

Tendons should never be sutured if the wound is already infected, if over twenty-four hours old, or even within twenty-four hours if in the presence of too much crushed or damaged tissue, or if the wound is too dirt-ground or badly contaminated; nor should the operation be done with poor hospital facilities or by an unskilled surgeon. It is preferable to leave a severed tendon undisturbed, so that it can be repaired later under good conditions than to do what will make the limb worse.

In recovering tendons for suture, a prevailing fault is to continue an incision upward along the tendon,—namely, to make the "pernicious median longitudinal incision" that cuts across natural creases and pulleys, and leaves a maximal length of adhesions along the tendon resulting in keloids, flexion contracture, loss of gliding surface, bowing forward of tendons, and immobility from adhesions. Instead, a small transverse incision higher up will suffice or a mid-lateral one in the finger. Care should be taken to avoid crossing flexion creases at a right angle or injuring small nerves.

Healing of Tendons

A clear conception of how a cut tendon behaves and heals helps in developing a method of suture. The healing is quite different when a tendon is cut in a sheath than when cut in paratenon formation, and different too when not sutured than when sutured. If cut in a sheath, the proximal end of a tendon retracts further than when cut in paratenon. In either case the distal end retracts only as far as it is drawn when the limb is extended. The tendon ends do not proliferate any more than to become rounded over and sealed by epitenon, and they remain free in the sheath. If infection develops, they become attached to the sheath and cicatricial.

When cut in paratenon and not sewed, each tendon end attempts to reach out to rejoin the other. Extensive proliferation of the paratenon, epitenon, and endotenon, or connective-tissue elements, reaches out like an expanded flame and attaches to anything it can. At first, it is tubular and jellylike, but connective-tissue cells quickly grow in, followed by tendon cells which furnish strength to the attachment. The whole mass then contracts. This blind effort often disables because the flexor

digitorum profundus is held from contracting by this attachment of one tendon, and so can no longer flex the other fingers completely. If instead the pseudopodia of one tendon end meet the other, the tendon mass contracts, cells guided by stress become arranged longitudinally, and the tendon becomes reestablished.

When tendons are sutured, the same rapidly growing fibroblastic proliferation joins the tendon ends as a fusiform splint in four days, and in two weeks is quite advanced, with connective-tissue cells, but without strength. Tendon cells commence to proliferate in four days and bridge the gap in two weeks. They furnish strength, so that in three weeks splints can be removed, and in four weeks the danger of breaking is over. There is much swelling in the first two weeks. After that the connective-tissue elements loosen around the tendon to allow movement. Healing of tendon in paratenon is more active than that within a sheath because of the surrounding paratenon and the greater vascularity. Paratenon and epitenon are to tendons what muscle and periosteum are to bones.

In the above repair process, there is naturally great tendency for the tendon at its juncture to grow fast to the surroundings, and this tendency is stimulated by all factors which provoke tissue reaction, including both infection and surgical trauma. The more elaborate the suture, the greater is the amount of foreign-body material; and the more necrosis which develops from the grasp of the suture, the worse will be the attachment. It is clear that to obtain a free-running tendon, healing should be as reactionless as possible.

The poorest results in tendon repair are of the flexor tendons in the narrow firm tunnel, or sheath, extending from the distal crease of the palm almost to the middle joint of the finger. Here the enclosing firm tunnel does not allow for the necessary swelling of repair, the blood supply is squeezed out, and the tendon within the tunnel undergoes necrosis. Resolution of this results in converting all the tunnel contents into a firm cicatrix. To forestall such a mishap the fascial tunnel should be split its length laterally to decompress, and maintain vascularity of the tendon during the stage of swelling.

It is customary to obtain good results by silk suture of tendons in the forearm where the surrounding tissue, if adherent, moves with the tendon. This is true in the dorsum of the hand where also tendons interdigitate; but in a flexor sheath in a finger, failure is inevitable unless every refinement of method and delicacy of technique is used.

Materials for Suturing Tendons

Of materials for suture, catgut is the least satisfactory. It is coarse and provokes so much reaction, sensitization and otherwise, that its use is followed by infection six times as often (O'Shea[1]) as that of silk. Silk No. 2, untreated, is in general use, but, though superior to catgut, it causes considerable cicatrix about itself. Fine stainless-steel wire, sizes No. 34 and No. 35, provokes the least reaction of all suture materials. It is

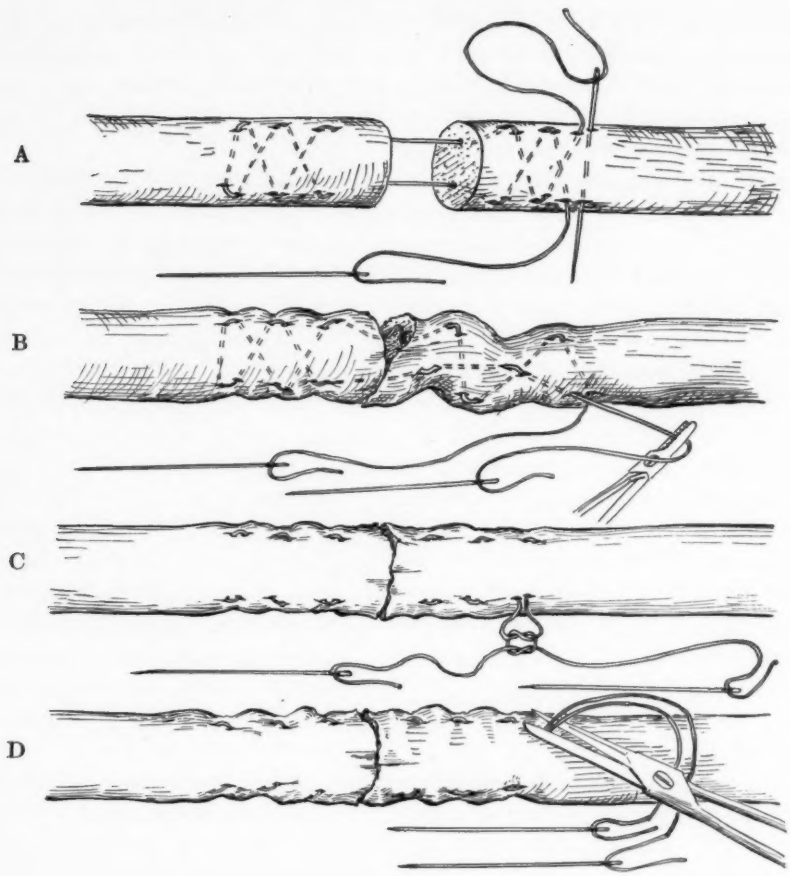

FIG. 1

Stitch for interval repair of tendons with stainless-steel wire. This stitch is too elaborate for primary suture. With No. 34 wire the juncture withstands a pull of five pounds. This stitch when uncovered months later usually shows less reaction in tendons than that from silk. Where it has been subjected to constant angular movement, as at the wrist, it may be found that the wire has broken into many tiny segments which do no harm, or, if not broken, that there are occasionally some signs of mechanical irritation. This is a point in favor of the removable wire suture which we now use routinely.

A. Placing the stitch.

B. All slack is removed by pulling on one wire at a time. The tendon slips on smooth wire and allows itself to be bunched up on the wires. This prevents separation of the tendon ends later.

C. A single knot, which sinks into the tendon when tied, is used remote from the suture line.

D. The ends of the wire are cut off, after being again passed through the tendon, so they will be left embedded within the tendon.

non-electrolytic and is apparently inert on the tissue in which it lies. It slides well through tissue and is, for its strength, finer than silk,—a suture with No. 34 will withstand a strain of five pounds. Great care should be used to avoid kinking the wire as this will cause it to break. For five years the author followed Babcock in using it for wound closure and for three years has used it for tendon suture. Tendons containing this

suture which were uncovered later, usually showed an even more normal appearance than those containing silk. In some cases where there had been continual movement over many months, the wire had fragmented into short segments, but these were not doing harm. Occasionally signs of mechanical irritation from the wire, due to movement, were found. A method has been developed of so placing the stitch in the tendon that it is removable in three weeks when the tendon will have physiologically united, thus leaving the tendon juncture free from any suture.

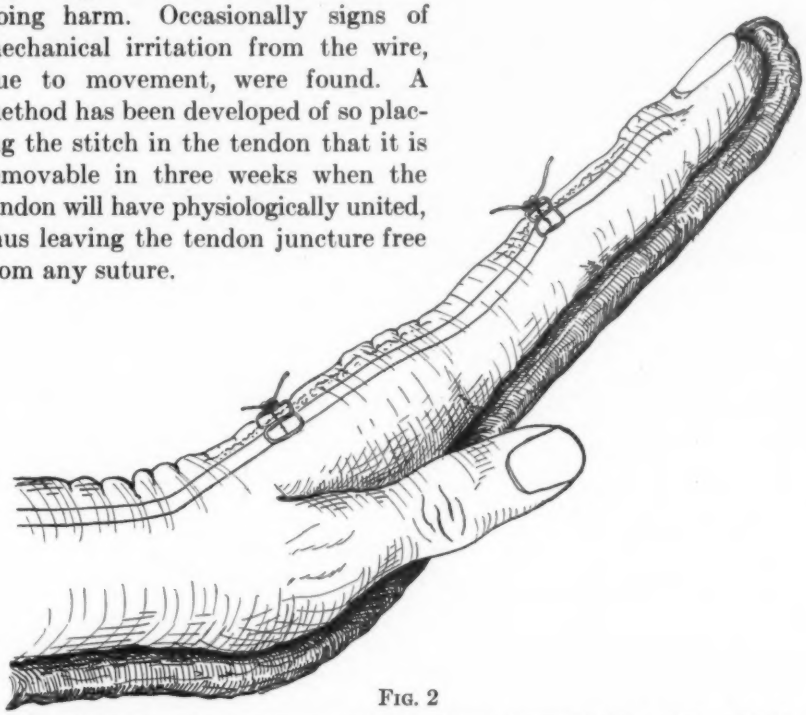

FIG. 2

For primary repair of extensor tendons a simple figure-of-eight stitch with No. 35 stainless-steel wire is used. One loop unites the tendon ends and the other the skin edges. The stitch is without strength and is merely for approximation of tendon ends. Splinting, as shown, prevents the tendon ends from pulling apart.

Type of Stitch

In the primary repair of tendons the stitch should be the simplest possible. In order to hold, it must cross some fibers for a spliced effect but this should be minimal. A single stitch placed longitudinally will split through on slight tension. A core suture (Fig. 1) is less likely to cause adhesions than one in which the suture is on the surface of the tendon. The smooth glistening epitenon surrounding the tendon should not be even scratched. The suture material should be fine and largely sunk out of sight into the tendon. All slack should first be withdrawn by pulling the sutures taut. Otherwise the tendon ends will pull apart somewhat during healing. There will be some central necrosis from this tightness, but it will eventually be replaced by tendon cells.

To prevent tendon ends from pulling apart more reliance should be placed on splinting, with joints flexed to relax the tendons, than on the strength of the suture (Fig. 2). In three weeks fairly strong physiological

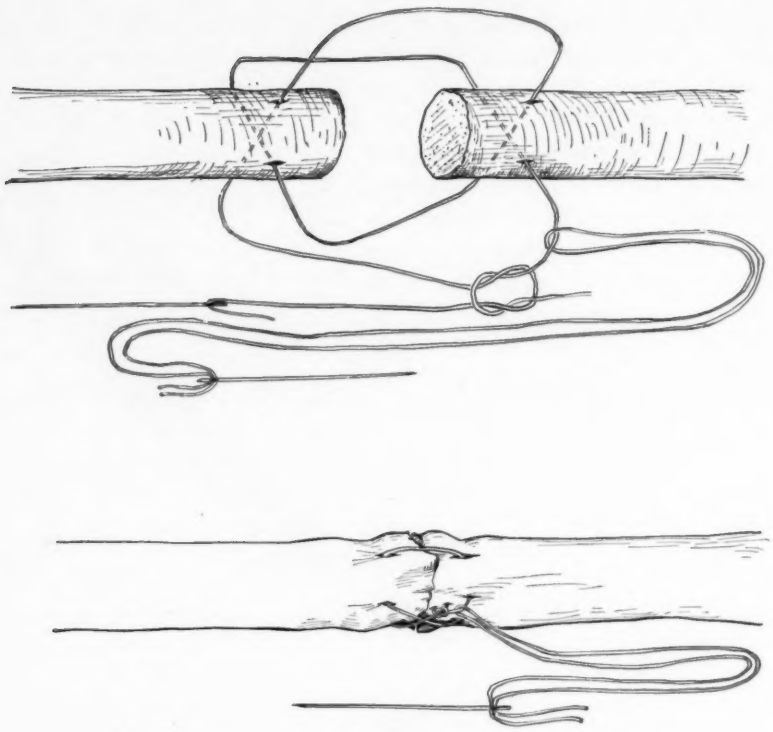

FIG. 3

Double right-angle stitch. This stitch is more than twice as strong as two simple through-and-through stitches because in each tendon end the stitch crosses diagonally a quadrant of tendon fibers. This displaces till somewhat on the bias as the stitch is pulled, all strands thus making for added strength. The four strands give a basket effect for better approximation of the tendon ends and divide the strain, so that as small as No. 35 wire can be used. The stitch is simple and quickly placed. To prevent breaking more reliance is placed on splinting the wrist in flexion to relax the flexor muscles than on the strength of this stitch. A pull-out wire is placed under the knot as shown, drawn out through the skin and left in place. If infection develops later, the stitch can be withdrawn as soon as the tendon unites, or if healing is *per primam,* the pull-out wire may be withdrawn.

union allows removal of the splint. Half circumference plaster of Paris is best for this. For flexor tendons it is molded on the dorsum of the hand and forearm with the wrist flexed, but with the digits free for some feeble action. For extensors the splint is placed on the volar aspect and holds the wrist and digits in dorsiflexion. The latter keeps extensor tendons so well relaxed that the only suture necessary is a simple figure-of-eight through the skin with one loop and through the tendon ends beneath with the other; this is kept *in situ* for three weeks. These extensor tendons so interdigitate that splinting alone often suffices, though the accuracy of union gained by one stitch is advisable if we gauge our results critically.

For flexor tendons in palm or forearm a useful quick stitch that holds fairly firmly is the "right-angle double" (Fig. 3). The needle is thrust straight through the tendon four millimeters from the end, repeating in

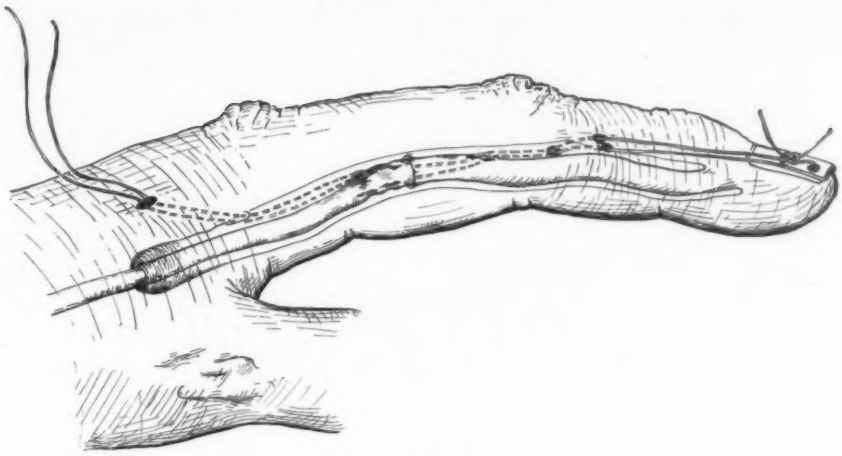

FIG. 4

Withdrawable stitch of stainless-steel wire. The stitch is made fast into the tendon end which is attached to the muscle and then anchored to the outside of the limb, in this case through the fingernail. A tiny stitch of blood-vessel silk or No. 40 stainless-steel wire approximates the tendon ends. The distal tendon end is passive and does not exert a pull. A pull-out wire is seen looped through the proximal loop of the stitch and brought out through the skin. Both the pull-out and the stitch wires are made to traverse the tendon sheath some distance before emerging through the skin. This allows some to-and-fro movement of the tendon in the sheath without causing parting of the tendon ends.

In three weeks, when the tendon is physiologically united, the stitch is withdrawn. After cutting off the stitch wires at the skin, the stitch is drawn out backwards by its loop by the pull-out wire.

the other tendon end as in the simple longitudinal stitch which will not hold. Another similar stitch is placed at a right angle to the first. The ends of the stainless-steel wire are then tied fairly tightly. No. 35 instead of No. 34 wire can be used, as four instead of two wires divide the strain. The four wires also hold the tendon ends cagelike and in better approximation. The strength of this suture is due to crossing the tendon fibers in two quadrants, but when used the tendon should be left in constant relaxation by keeping the wrist flexed by a plaster-of-Paris dorsal splint, as the juncture is not strong. These sutures can be placed so they may withdrawn or not at will.

Withdrawable Tendon Sutures

Convinced that complete absence of suture, when after three weeks the tendon has united, is most favorable for a free-running tendon, a method has been developed by which the suture can be withdrawn (Fig. 4). After a tendon is sutured, only the end of the tendon attached to the muscle exerts a pull. Therefore, the suture is spliced securely in this end and the two ends are passed some distance down through the center of the distal tendon and on out through the skin. By pulling upon these sutures from the outside and anchoring them securely, the proximal end can be drawn down until the tendon ends are held approxi-

FIG. 5-A

FIG. 5-B

G. H. Result in case of primary repair of severed flexor tendons in little finger sutured in the proximal segment, a part of the finger notorious for poor results. Removable stainless-steel wire No. 34 was used by the method shown in Fig. 4.

mated. Only one tiny stitch of blood-vessel silk or hairlike wire is then necessary for exact approximation of the tendon ends.

For the later withdrawal of the stitch a pull-out wire is left in place which will draw the stitch out backwards by its loop whenever the two stitch wires are cut. Both ends of the pull-out wire, after being placed through the loop of the stitch in the proximal tendon end, are threaded on a needle and passed up through the sheath some distance and out

FIG. 6-A

FIG. 6-B

E. M. Another case of primary repair of severed flexor tendons.

through the skin. The play of the wires through the sheath will allow a little movement in the tendon without strain on the juncture.

Tension is relieved by flexing the joints. The stitch wires can be anchored to the outside of the limb by passing them through adhesive plaster applied to the skin and then fastening them through a button, by "split shot" or to a loop of adhesive plaster over the end of the finger, or by tying the wire ends through a perforation in the fingernail. If any resistance is encountered when withdrawing the stitch by the pull-out wire, tension may be applied by fastening a light rubber band to the wire. At the next dressing the stitch will be out. The bend of the pull-out wire, where it loops through the stitch, should be pinched sharp, so that it will not encircle tissue.

If the right-angle double stitch is used, it can be made optionally withdrawable by merely laying a pull-out wire across under the knot as it is being tied. The two ends of the pull-out wire are then threaded on a

needle and brought out through the skin. If infection occurs, the stitch can be withdrawn, but if it does not, the pull-out wire alone is withdrawn after cutting off one of its ends just inside the skin.

General Considerations

Block anaesthesia is generally used with the ischaemia of a tourniquet. An injection is made of five cubic centimeters of 2-per-cent. novocain plus adrenalin in the ulnar nerve at the elbow, the median nerve above the wrist, and the branches of the radial nerve as they reach the superficial fascia. If the case is extensive, general anaesthesia is preferred.

It is important for the surgeon to be comfortably seated and to work across an arm board with an assistant. The arm board is made steady on a firm base and the patient's hand is held immobile by the assistant who presents it properly to the operator's line of vision under a bright but not a hot light.

If in a finger the flexor sublimis tendon is cut, it need not be sutured, but if both flexors are cut only the profundus should be repaired, and the sublimis should be removed. If both tendons are sutured, they will adhere to each other and thus act on the middle joint alone, but not on the distal joint. The profundus tendon sutured in the middle segment of the finger may yield a fair result, as it need pull through but one joint and not two as in the proximal segment. Also, if it adheres it may later be freed with some advantage. Flexor profundus tendons are essential for good function, but sublimis tendons are not so important. They help the muscle balance of the fingers and furnish the final tight clenching. Good results are obtained in ruptured insertions of extensor tendons if the finger is splinted early in hyperextension of the distal and flexion of the middle joints. Suturing is unnecessary in fresh cases.

Flexor severance in the proximal segment of a finger is not only the most common tendon injury, but is followed by the poorest results. When the problem of repairing a flexor tendon at this site is solved, the repair of all tendons will be comparatively easy.

CONCLUSIONS

Primary repair of tendons in open wounds is dependent upon obtaining primary healing in the wounds in general.

Essentials are prompt operation, thorough débridement, covering over all vulnerable tissues, and adequate after-treatment, consisting of splinting, elevation, and dryness.

The technique of tendon suturing is based on physiological healing.

Stainless-steel wire is superior as a suture material.

Irritation from suture is terminated by withdrawing the suture from the tendon in three weeks.

The fascial tunnel or sheath in the proximal segment of a finger should be split laterally when repairing a tendon within it.

1. O'Shea, M. C.: Severed Tendons and Nerves of the Hand and Forearm. Ann. Surg., CV, 228, 1937.

SUBFASCIAL HEMATOMA AS A COMPLICATION OF CRUSHING INJURIES TO THE FOOT

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Due to the form of the human foot and its manner of functioning, the forefoot is constantly exposed to crushing injury. Heavy objects may fall upon the unprotected feet of laborers, while the feet of pedestrians may be run over by vehicles or caught in sliding doors or escalators. Marked subfascial hemorrhage on the dorsum of the foot usually accompanies such injuries. Local circulatory obstruction of varying degree is produced immediately. Unless this subfascial tension is promptly relieved, permanent damage is done to the soft structures of the forefoot.

This report is based upon four such cases. Three were acute and were given immediate attention, whereas one patient was admitted twenty-four hours after injury. This last-named patient developed a relatively large trophic ulcer on the dorsal surface of the forefoot. Because of the delay in admission, the area had become devitalized by pressure of the underlying hematoma.

From a study of the anatomy it is evident that crushing injury to the foot may produce serious complications, because of the vulnerability of the blood vessels. The horizontal and perforating arteries, for the most part, lie beneath the dorsal fascia. The deep veins communicate with the saphenous system by means of perforating branches through this fascia. Passing transversely across the anterior aspect of the ankle are two well-defined bands of tissue, the cruciate and transverse ligaments (Fig. 1). The strips are continuous below with the dorsal fascia.

Following trauma, these ligaments, particularly the cruciate, may become constricting bands to produce circulatory obstruction. Immediately after the injury there is local vasodilatation and a bulging out of the fascia due to the confined subfascial hemorrhage. Because the dorsal fascia blends laterally with the thick plantar fascia, this hemorrhage does not pass readily into the sole of the foot. The venous flow from the toes is obstructed first, and, as noted in Case 1, the condition is rapidly progressive. Another factor is that most persons so injured are brought to the hospital with the fractured foot dependent. If a traumatized foot is elevated promptly after injury, a complete circulatory block may be prevented. In violent crushing injury, however, the arterial hemorrhage rapidly fills the subfascial space and the distal forefoot quickly becomes cold and cyanotic (Fig. 2). Marked oedema, which obscures the true condition, later appears in the loose subcutaneous tissue.

To determine, experimentally, the rôles of the cruciate and transverse

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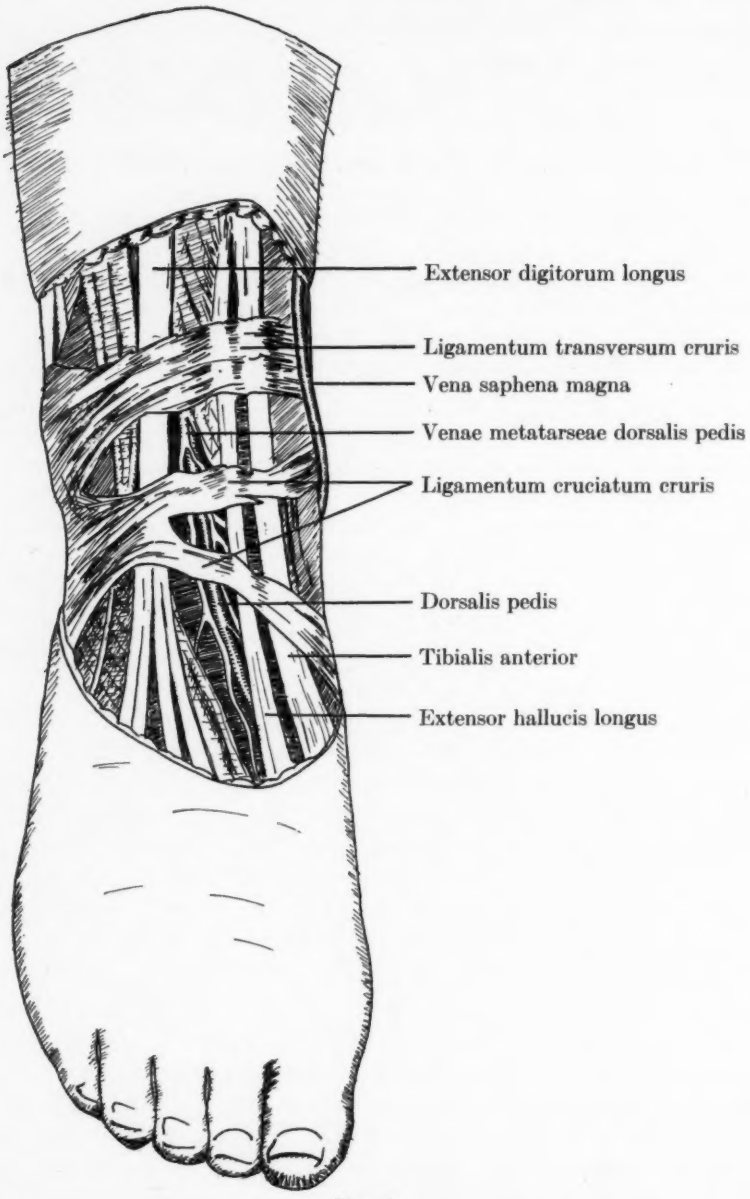

FIG. 1

Cruciate and transverse ligaments. (Drawn by Merrill Leinbach from Cunningham's Manual of Practical Anatomy, Ed. 7, Vol. I, p. 349. Edinburgh, Glasgow, and London, Henry Frowde and Hodder & Stoughton, 1919.)

ligaments in subfascial hemorrhage, six feet from unembalmed cadavera were injected with a solution of black dye and physiologically normal saline. The evidence obtained was more indicative than conclusive. Injections varying in amount from 50 to 200 cubic centimeters were made beneath the deep fascia on the dorsum of the foot and into the dorsal

interosseous spaces. Dissection was done from five to thirty minutes later with exposure of the fascia, tendon sheaths, and the cruciate and transverse ligaments. Particular attention was given to the cruciate ligament, and the belief that this ligament would act as a confining band in deep fascial injections was confirmed in every instance. In ten to fifteen minutes, however, a small amount of the dye solution passed beneath the ligament to continue upward to the transverse ligament where it was delayed still further. In two of the feet injections of 150 and 200 cubic centimeters were made after the fascia and the ligaments had been exposed. The dorsal fascia ballooned out considerably up to the cruciate ligament which was put under tension. When this ligament was sectioned transversely in the mid-portion, its ends separated 3.5 centimeters in one foot and 4.6 centimeters in the other. It was suggested that a dyed acacia solution be used rather than the saline. This method was employed in two additional feet with identical results. In no case did the fluid pass beyond the edge of the sole and none extended beyond the webs of the toes.

Conclusions from these injections into unliving tissue are made with reservations since the actual changes occurring in living traumatized feet cannot be produced.

Treatment depends largely on the severity of the case. The general condition of the foot should be considered first rather than the fracture. If deformity due to the latter is causing additional venous congestion, reduction should be done and retained by skeletal traction or pillow splint. If the patient is seen soon after the injury and only a moderate venous obstruction is present, elevation of the foot, application

FIG. 2

Case 3. Crushed foot showing swelling and cyanosis.

of ice packs, and gentle centripetal massage of the foot and ankle should be tried. In many of these cases such treatment is adequate. In severe cases the hematoma should be released at once by multiple incisions through the fascia. Soft rubber drains should be inserted and the wound left open. If this is not effective, then sectioning of the cruciate and transverse ligaments must be considered. Aspiration of the hematoma with a large-bore needle usually gives only temporary relief (See Cases 2 and 3).

Metatarsal fractures are usually found following such trauma, but soft-tissue damage alone may produce an identical clinical picture. The entire forefoot becomes swollen and firm. The skin over the dorsum is stretched tight and is cyanotic. The toes are cold and insensitive to pain and temperature changes; the dorsalis pedis is not palpable. This injury, to some degree, is comparable to the subfascial hemorrhage in the forearm preceding Volkmann's contracture. The resultant muscle damage on the dorsum of the foot is negligible, however, as only the extensor digitorum brevis and dorsal interossei are exposed. As far as could be determined, these muscles were not affected in any of the four cases.

CASE REPORTS

Case 1. White female, aged seventy-two years, had her left foot run over by an automobile twenty minutes before admission to the hospital. There was a tense fluctuant swelling over the dorsum of the foot; pulsation of the dorsalis pedis was absent. There was a definitely depressed line about four centimeters in width across the dorsum of the foot at about the level of the cruciate ligament. The toes were cold, bluish-black, and did not react to sensory stimuli. Roentgenograms showed a fracture of the fifth metatarsal bone without displacement. For one and one-half hours the patient refused treatment while the area of cyanosis and anaesthesia spread up from the webs of the toes to the mid-tarsal joint. All the swelling remained confined to the anterior and anterolateral portions of the foot. The plantar area was relatively normal in appearance.

Two two-inch longitudinal incisions were then made down through the deep fascia proximal to the first and third metatarsal heads. Several large soft blood clots were removed and about sixty cubic centimeters of dark blood was evacuated. Soft rubber drains were inserted, the wounds were left open, and the foot was elevated beneath a light cradle. Immediate improvement was noted, and twelve hours later the foot remained only moderately swollen and discolored. Six days later the color and sensitivity of the foot were practically normal. Pulsations of the dorsalis pedis artery, however, did not return.

Case 2. White boy, aged thirteen, was struck by an automobile which ran over his left foot. He was brought to the hospital at once with a compound posterolateral dislocation of the ankle and with the entire forefoot cold, cyanotic, and insensitive. The dorsalis pedis artery was not perceptible. He was in moderate shock. As the dislocation appeared to be causing a circulatory block, the wound was scrubbed with green soap and the dislocation was reduced. As the circulation did not return, and as there was evidence of a subfascial hematoma, about thirty cubic centimeters of dark blood was aspirated. The circulation and sensitivity returned almost immediately to the toes and to the dorsal forefoot. Roentgenograms showed a transverse fracture of the mid left tibia and fractures of the second, third, fourth, and fifth left metatarsals. Two hours later the toes and forefoot were again cold, cyanotic, and anaesthetic. Two longitudinal incisions were made through the dorsal fascia and about fifty cubic centimeters of dark blood with a few soft clots was removed. Drains were inserted and the wounds were not closed. The leg was elevated beneath a light cradle and the drains were removed twenty-fours hours later. The foot appeared normal the next day and circulation to it remained unimpaired throughout convalescence.

Case 3. White male, aged thirty-two, was injured while unloading lumber from a truck. Several large timbers fell upon his left leg and foot. On admission shortly afterward the dorsal fascia of the foot was tense because of deep hemorrhage and the ankle joint bulged with fluid. He complained of numbness over the entire foot. Roentgenograms of the foot showed fractures, with only slight displacement, of the first cuneiform, third and fifth metatarsals, and the calcaneum. Eighty cubic centimeters of blood was aspirated from the dorsal subfascial space and the ankle joint. The circulation improved to such an extent that a plaster casing was applied and the extremity elevated. The patient soon complained of severe pain, and upon bivalving the plaster the foot was found to be discolored and cold. Multiple releasing incisions were considered but not done. The leg was elevated higher and a heat cradle placed over it. The circulation showed some improvement, but in twelve hours multiple large fracture blisters appeared over the foot and ankle. Despite later intensive physical therapy a marked brawny swelling persisted for a period of over three months.

It is believed that multiple deep-fascial incisions should have been made in this case. According to Mock [1] such a failure to relieve immediately the subfascial swelling results eventually in a painful foot accompanied by cyanosis, clammy skin, and thickened swollen tissues. The convalescent period is extended to an extreme degree and the end result is apt to be disappointing. He calls such a result the "congealed" foot.

Case 4. Colored boy, aged six, was admitted to the hospital twenty-four hours after his left foot was run over by an automobile. The circulation to the third toe was completely obstructed and he had moderately severe swelling of the forefoot. The toe could be moved actively, but was insensitive to stimuli. Twelve hours after admission a hematoma was removed from beneath the fascia by longitudinal incision. The skin and subcutaneous tissue, as seen at the operation, appeared grayish in color, and devitalized. Both the circulation and sensitivity returned to the third toe within twelve hours. Two days later, however, an area the size of a silver half dollar sloughed out. The ulcer required a period of several weeks to fill in with granulation tissue.

SUMMARY AND CONCLUSIONS

1. Subfascial hemorrhage of the dorsum of the foot frequently complicates metatarsal fractures and soft-tissue damage caused by crushing trauma.

2. The cruciate ligament of the foot may act as a constricting band in subfascial swelling. This condition was produced in eight feet of cadavera injected with dye solutions.

3. Immediate multiple incisions to release and to remove the subfascial hematoma is the treatment indicated in severe cases.

4. Release by transverse section of the cruciate and transverse ligaments of the foot and ankle should be considered in persistent circulatory block.

5. Delayed or inadequate treatment results in the "congealed" foot or in necrosis of the soft tissues of the dorsal forefoot and toes.

Acknowledgment is made for the helpful suggestions of Kenneth C. Farnsworth, M.D., of the Department of Anatomy, Harvard Medical School, in the making of this study.

1. Mock, H. E.; Pemberton, Ralph; and Coulter, J. S.: Principles and Practice of Physical Therapy. Vol. II, pp. 111 and 119. Hagerstown, Maryland, W. F. Prior Co., Inc., 1934.

SLIPPING OF THE UPPER FEMORAL EPIPHYSIS

End Results After Conservative Treatment

BY M. FORRESTER-BROWN, M.S., M.D.(LOND.), BATH, ENGLAND

It is the desire of the author to report in this paper the results of treatment for slipping of the upper femoral epiphysis by a conservative method. The study is based on her experience with twenty-two cases treated at the Bath and Wessex Orthopaedic Hospital and followed for a number of years. No claim is made that this treatment by manipulation produces anatomical reduction or perfect function; however, it has given sufficiently good functional results to warrant reporting, particularly in view of the fact that operative methods of treatment do not bring about 100-per-cent. "cures", while some involve a certain risk to life and others may damage the articular cartilage. Moreover, it is not possible to have all cases cared for in the larger centers where more complete equipment is available.

It is impossible to assess the value or the results of a method without analyzing some of the features of the cases treated and the nature of the disease. Accordingly, a brief review is presented of the findings in the series of cases under consideration.

Displacement in adolescents has many features in common with fracture of the neck of the femur in the aged, but differs in that there is usually no actual separation of the fragments, but only a bending and twisting of the cartilage, and particularly a turning of the head backward on the neck, which often makes reposition difficult. When anatomical reposition is shown roentgenographically, the problem of fixation is not only one of maintaining exact contact for a matter of weeks, until new bone bridges the gap, but also of reducing stress on the cartilage of the weak neck until growth and metabolic changes have strengthened it sufficiently for weight-bearing and the rough usage of a normal adolescent's life.

It should be emphasized that the object of treatment should be to obtain a joint whose range of movement, however limited, is through the most valuable or important arcs, rather than perfect reduction as shown by the x-ray, with a flexed, abducted, or adducted limb. It has been the experience of the writer that, with the technique to be described, there has been a tendency toward a slight increase in the range of motion as the years have passed; whereas cases have been reported where good motion was obtained by more radical measures, but stiffness has gradually developed and good position has not been maintained.

FACTORS WHICH INFLUENCE THE RESULTS OF TREATMENT

Metabolic Disturbances

Endocrine abnormality was evident in nineteen of the author's twenty-two cases, with the usual physical signs of excessive weight and

height. Some showed increased sugar tolerance, and most of these improved when treated by thyroid, but in some, benefit was obtained only after thyroid had been given alternately with fortnightly courses of injections of pituitary whole gland. This treatment was supplemented by measures to improve the calcium metabolism and to reduce the weight imposed on the weaker structures. All of the patients were treated in the open air, with heliotherapy, and a diet rich in vitamins.

Of three thin patients, one had recovered from a tuberculous knee, one had a rheumatic heart, and the other had spasmodic flat-foot.

Latent Sepsis

There were three patients with fibrositis and joint stiffness in other parts of the body, three with septic tonsils which required removal, and one with goiter of the adolescent type. Possibly these factors are only in a proportion normal to the child population.

The factor of sepsis is important, because, if the patient should develop a tendency to joint stiffness, this might be aggravated by forced manipulation of the joint or by operation in and around the joint.

Vulnerability of Growing Tissues to Strain in Adolescence

The influence of growth is one of marked importance. In adolescents, if weight-bearing can be brought into a good mechanical condition, the bone will then reconstruct itself and tend to restore its femoral neck and shaft. This tendency is shown very distinctly and prominently after Lorenz's bifurcation osteotomy for congenital dislocation of the hip, and the same restorative forces are at work in the healing of any disease of the femoral neck and head, provided unfavorable strain can be abolished.

There is no quick cure for a slipped epiphysis. With the head once in favorable position for healing, the position must be maintained for a long time, which may not be the same in any two cases. In the author's opinion, it is not until bony trabeculae can be seen unmistakably to cross the epiphyseal line from the neck to the head, that safety against relapse is assured. Union of the upper epiphysis on the injured side tends to occur before it does on the sound side, although it must not be forgotten that cases often are bilateral.

Although this is in a sense a growth disease, it does not lead to any appreciable shortening. One-half inch was the average in the author's cases. There was shortening of over an inch in only one patient, and that was a boy with a rheumatic heart who grew rapidly while the leg was kept in fixation.

Age at Onset

Sixteen of the twenty-two cases occurred between the ages of twelve and fifteen years. The youngest patients were two overgrown girls of ten years; two were eleven; and two extremely tall boys were sixteen and seventeen respectively. It might be thought that the younger cases

would show the best results; whereas, of the four hips which ultimately became stiff, two were those of girls of ten and eleven years, treated by different methods, and two were of girls of fourteen.

Interval Between Onset of Symptoms and Treatment

The length of time between the onset of symptoms and manipulation might be thought to have a great influence on the result, but this is not borne out by the results in the cases under consideration.

It is extremely difficult to know what date to consider as the beginning of the disease, because in practically every case there had been months of vague pain in the groin or down the thigh of the affected side, often with intermittent limping, before the patient was brought to the doctor. In most cases there was the usual history of some sudden but rather trivial injury. This suggests that the softening process in the bone gives rise to vague discomfort before it weakens the tissues enough for slight trauma to cause displacement.

Bone-Softening

The existence of generalized and persistent bone-softening in these cases is demonstrated by the frequency of severe knock-knee as a complication, much aggravated if there is spasm of the hip adductors. Ten patients, both girls and boys, showed this complication, and it varied in severity from a gap of one and one-half inches to four and one-half inches between the malleoli, when the patient was seated. When plaster is being applied to the hip, great care should be taken to see that the assistant who holds the limbs corrects this deformity as much as possible; if one is not careful, his zeal to keep the hip abducted results in his pulling the tibia outward on the femur! The presence of knock-knee is one of the factors that make treatment without abduction of the hip desirable.

TECHNIQUE

In the earlier cases it was considered that a preliminary loosening of the fragments was necessary, and in seven cases Kirschner wire was applied, with traction on the femur by weights of from fifteen to twenty pounds, for three weeks. In four of these cases, the wire was removed under general anaesthesia and the hip manipulated and placed in plaster. The other three were treated by splinting, as the external rotation and flexion of the hip had become corrected under the traction. The x-ray results of these cases were disappointing, as the only effect of the heavy weights had been to pull the hip below its normal level in the acetabulum without affecting the relation of the head to the neck. In view of the experience with these earlier cases, skeletal traction was discarded, although there may be exceptional cases which would benefit by it. In the last eleven cases of the series the writer has used the following simple technique.

An anaesthetic is given in almost all cases. With the method of Leadbetter always kept in mind, an attempt is made to bring the hip into

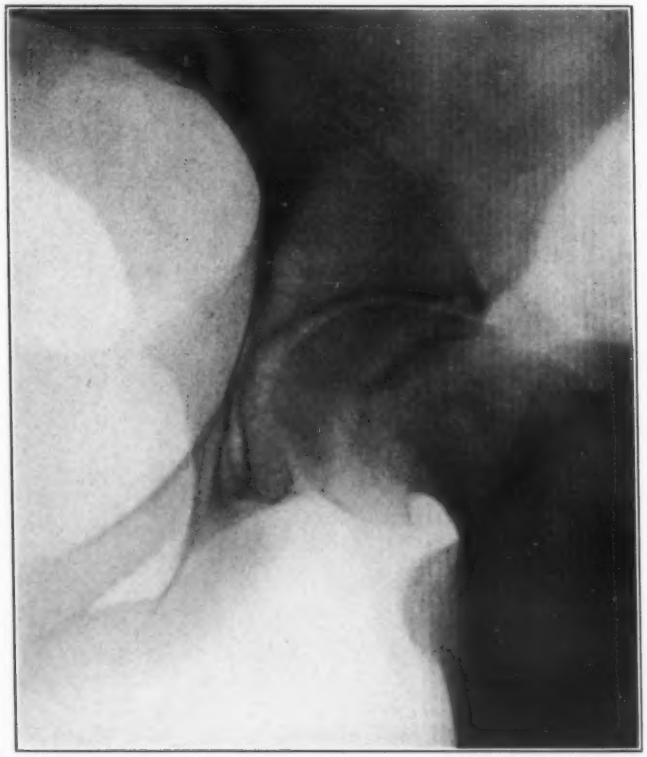

FIG. 1-A

M. S. Female, aged fifteen years.

Roentgenogram taken June 3, 1935, one month after onset of pain and limp. Thirteen months before, she had fallen on polished floor.

After treatment by piano-wire traction for three weeks, the hip was placed in plaster for three months, in internal rotation with no abduction. Girl kept in bed for two weeks, until muscles were good; then allowed up but used walking caliper for three months.

FIG. 1-B

M. S. Roentgenogram taken October 12, 1937, two years after treatment.

Patient seen early in 1940. She is now twenty and has been working as a dressmaker and mannequin. Her present range of motion shows 70 degrees of flexion in each hip, full extension, little rotation from neutral, adduction equal on both sides, but abduction not quite equal on both sides.

full internal rotation (an amount at least equal to the maximum obtained on the sound side), but this can seldom be accomplished except by first flexing and then adducting the hip. After this it is usually possible to internally rotate the limb into extension without losing the internal rotation, and gentle, steady pressure may be necessary to prevent its springing back into external rotation. This position must be held until the limb lies naturally in internal rotation. A plaster spica is then applied with the limb in this position, maintaining definite extension with a minimum of abduction. The foot is included in the plaster. When the plaster has dried, the patient is allowed to walk and to return home for three months, at which time he is readmitted for x-rays and observation. If sufficient abduction is present, a lateral roentgenogram taken with the limb in "frog" position is valuable to show the relation of the head to the neck, with the sound side for comparison. Until the epiphyseal line is crossed by trabeculae, it is deemed that slipping may recur, and a series of plasters are reapplied until this is obtained.

RESULTS

Eleven patients, six girls and five boys, have been treated in the manner described, at periods of from one to three months after onset of symptoms in six cases and from six to twelve months in five cases.

Serial x-rays of these cases every few months for years after the manipulation show interesting and progressive reconstruction of the upper end of the femur, by which the line of weight-bearing is gradually deflected into normal lines with increased density of new trabeculae, which crystallize in the lines of stress and absorption of parts. This crystallization is not so evident in the parts which no longer function, like the lowest quadrant of the head. The upper border of the neck resumes its normal line, concave downward. The neck does not usually become as long as that on the sound side, nor is the head quite normal in shape, and it is possible that investigation of these cases twenty or thirty years hence will show an undue proportion of osteo-arthritics. That may be an inevitable sequel of the disease, as of so many abnormalities of the hip.

In all cases full extension of the hip and internal rotation to neutral were maintained; in only one was internal rotation increased even though all had been fixed in plaster well beyond neutral. All patients walked with a normal attitude; all had some limitation of flexion, but were able to sit without discomfort. Nine had limitation of abduction and adduction, and of external rotation; only one had a full range of motion in all directions.

As regards working capacity, seven (three girls and four boys) had been at full work in the open labor market for several years when recently seen,—some as engineers, some in shops, in factories, and in domestic work, and one in a timber yard. Two were attending ordinary schools; two had moved to other areas while still of school age, and attendance is unknown; two females were only helping at home.

FIG. 2-A

D. N. Male, aged fourteen years.

Roentgenogram taken on admission, July 31, 1934, four months after onset of limp and pain in hip.

After manipulation into internal rotation under anaesthesia, the hip was put into plaster spica (in which the foot was included) for three weeks. The patient was kept prone and treated by massage. He used a walking caliper for several months.

FIG. 2-B

D. N. Roentgenogram taken June 27, 1939, five years after manipulation.

He has been working as an electrical engineer for several years and was recently accepted for the army. He has full range of motion in the hip except that there is no internal rotation beyond neutral.

One boy of twelve years was admitted to the Hospital with the characteristic deformity and endocrine defects, but the x-ray showed no displacement, only an irregular epiphyseal line, and his symptoms cleared up with recumbency and massage, without splinting. When he was allowed to go home in one month, he had full range of movement which was still maintained when seen eighteen months later. This is the ideal result and suggests that in this disease, as in so many others, the essence of good treatment is early diagnosis, and that "sprains of the hip", like those of other joints, should be x-rayed at once and treated as if serious.

Unless such early diagnosis is possible, the writer contends that in properly selected cases the simple manipulation and persistent plaster treatment outlined will produce hip joints with good function, and that the results compare favorably with those of methods more difficult to carry out and more dangerous to life and limb.

MUSCLE TESTING AROUND THE SHOULDER GIRDLE

A Study of the Function of Shoulder-Blade Fixators in Seventeen Cases of Shoulder Paralysis

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Because of the great number of muscles which directly or indirectly participate in the movements of the shoulder girdle and the arm, it is very difficult to determine the specific function of each muscle. When studying origin and insertion of muscles on a skeleton, or when dissecting muscles on a cadaver, the exact anatomical position can be learned, and conclusions may be drawn as to their probable function. However, it has been shown repeatedly that, although a muscle may have a perfectly good anatomical position for carrying out a movement, it often lies inactive when that movement is performed. The function of muscles, therefore, must be studied primarily on the living human body. Careful observation and palpation of muscles on the normal individual yield a great deal of information; but for a thorough understanding of muscle action additional studies of paralysis cases are imperative. If the paralysis involves a number of adjacent muscles, as is usually the case in poliomyelitis, no definite conclusions can be drawn as to individual muscle action. For this, patients must be selected for study who have an isolated paralysis of one or two muscles, with all other muscles intact.

FIG. 1

From the standpoint of etiology, the seventeen cases

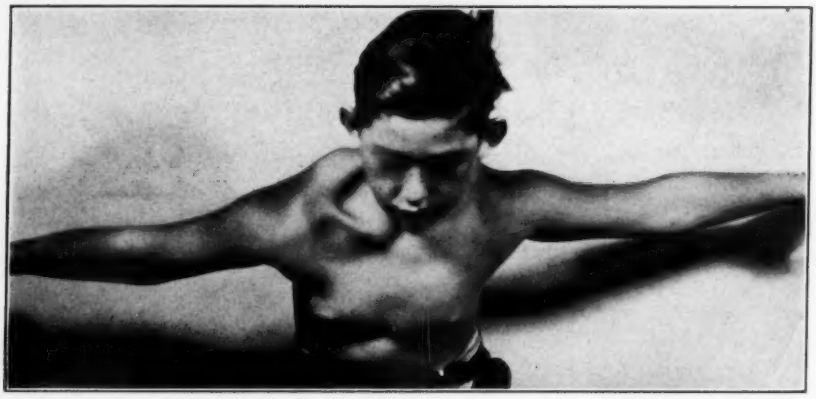

FIG. 2

chosen for the study are classified as follows: (1) injury or disease to the long thoracic nerve (one case), (2) injury to the spinal accessory nerve (four cases), (3) congenital muscular defects (seven cases), (4) muscle dystrophy (four cases), and (5) poliomyelitis (one case). These unusual types of shoulder palsy affect those muscles which connect the scapula and the clavicle with the main skeleton. These muscles have to do primarily with the movements of the scapula on the rib cage. They anchor the scapula to the trunk in all movements of the arm, rotating the scapula in whatever direction is required for the motion. Without these shoulder-blade fixators there is no control of the scapula, and the muscles acting on the arm become very inefficient.

The purpose of this paper is twofold: (1) To discuss positions and movements which to the greatest possible extent bring out isolated action of the shoulder-blade fixators and which make palpation of each muscle possible; and (2) to show by actual examples the functional disturbances which accompany isolated paralysis of these muscles.

To avoid misunderstanding, the following definitions are given:

Upward rotation of the scapula will mean that the glenoid cavity is turned upward while the inferior angle moves away from the spinal column.

Downward rotation of the scapula will mean the reverse movement of the above. In complete downward rotation the inferior angle of the scapula is closer to the spinal column than the superior; bilaterally, the vertebral borders of the scapulae form the letter V (Fig. 1).

Tipping forward of the scapula, that the coracoid process is lowered anteriorly while the inferior angle protrudes backward.

Trapezius

The various portions of the trapezius can easily be seen in normal subjects because they lie immediately under the skin. To bring out clearly the action of all parts of the trapezius simultaneously, one of the following tests is suggested:

1. Subject is in a sitting position with the trunk inclined forward

and the spine in a straight line; the arms are raised to a horizontal position with the elbows either flexed or extended (Fig. 2).

2. Subject lies face down with the arms abducted 90 degrees, the elbows either flexed or extended, and the arms raised off the floor.

3. Subject is in a sitting position with the hands on top of the head. The elbows are pushed backward against resistance (Fig. 3).

Any of these positions will reveal whether there is an absence or weakness of the trapezius as a whole or of one of its parts. The third test brings out most clearly the individual parts of the muscle. If any fibers of the lower and middle portion are present, they will contract vigorously when the subject attempts to push the elbows backward (Fig. 3). If the deltoid is weak, the resistance must be applied directly to the scapula.

When the whole trapezius is missing and the subject stands with arms hanging relaxed at sides, the scapula assumes a downward rotated position. The weight of the scapula and the arm, no longer supported by the trapezius, pulls down on the distal end of the clavicle, causing a considerable leverage action on the sternoclavicular joint. A subluxation of this joint is frequently the result (Fig. 4). An anterior view of a subject with involvement of the lower and the middle portions of the trapezius often shows a fold in the skin between the arm and the rib cage anteriorly to the axilla on the affected side. This skin fold is more pronounced in

FIG. 3

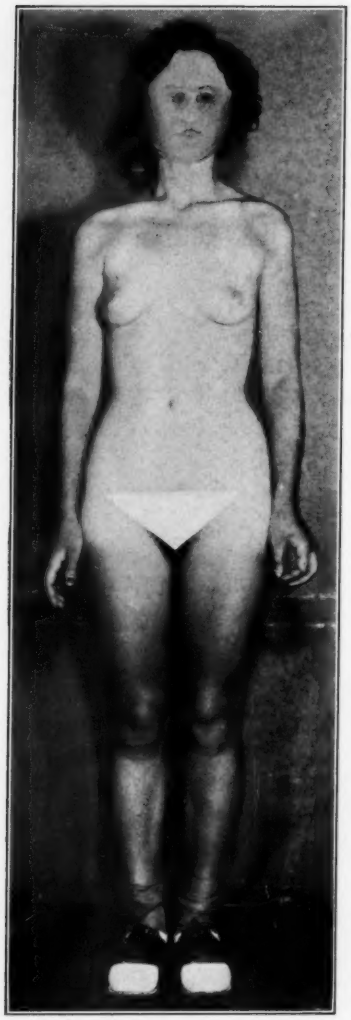

FIG. 4

subjects with well-developed adipose tissue. When the lower portion alone is missing, the skin fold may not be very noticeable, but there is always a tipping forward of the scapula, which causes the inferior angle to protrude. However, one must be careful not to conclude that all children with a protrusion of the inferior angle have an absence of the lower trapezius. This condition, when seen bilaterally, is most often a sign of poor posture; when present unilaterally, a lateral curvature of the spine may be the cause.

To test the upper portion of the trapezius separately, the subject is told to draw the shoulder toward the ear while slightly abducting the arm. In normal individuals, elevation of the shoulder girdle is accomplished through the combined action of the upper trapezius, the levator scapulae, and the rhomboids. The upward rotary action of the former muscle is counteracted by the downward rotary action of the two latter muscles. If the arm is somewhat abducted while the shoulder is raised, the rhomboids and the levator scapulae tend to relax, and the movement is performed mainly by the upper trapezius. Vice versa, the action of the levator scapulae is best studied when the shoulder is raised while the arm is adducted. By having the subject alternate these two movements (shoulder elevation with arm in abduction and the same movement with the arm in adduction), one can, through palpation, differentiate fairly well between these two muscles. If resistance is offered to the movement, both muscles contract in either case.

Absence of the upper portion of the trapezius causes a characteristic change in the outline of the neck and the shoulder which is particularly noticeable when the shoulder girdle is elevated. On such cases the levator scapulae can be seen in action directly under the skin. The posterior border of the distal end of the clavicle likewise can be seen, since it is not concealed by the trapezius, and the supraspinous fossa can easily be palpated (Fig. 5).

When the lower and middle portions are weak, the scapula cannot be

held in position when Test 1 is given (Fig. 2), and no resistance can be given in Test 3. If some fibers are still functioning, they can be seen contracting strongly (Fig. 3).

In all cases which were observed, partial or complete absence of the trapezius caused very little, if any, functional disturbance. Most subjects were unaware of any weakness and all movements could be performed without difficulty. The girl shown in Figure 3 had a complete absence of the upper trapezius, yet she displayed no difficulty in elevating the shoulder girdle. Surprisingly enough, when resistance was applied to this motion, it was found that the affected side was as strong as the unaffected. This girl had an almost complete absence of the middle and the lower portions as well. There was a very slight disturbance in the scapulohumeral rhythm, but the arm could be raised over the head without difficulty. Since the trapezius defect in this case was of long standing (possibly congenital), the girl had learned to compensate for the missing muscle with great success. The levator scapulae had taken over the function of the upper portion of the trapezius and the serratus magnus was able, without the aid of the trapezius, to perform the upward rotary movement of the scapula. Loss of the lower portion alone caused practically no functional disturbance. On the whole, the observation seems to indicate that the trapezius muscle is of less functional importance than is usually believed.

FIG. 5

Serratus Magnus

The anatomical position and shape of the serratus magnus makes observation and palpation of this muscle rather difficult. On thin subjects some of the lower digitations of the muscle can be seen at their origin on the ribs, but when much adipose tissue is present the muscle can be palpated only with difficulty. Pushing the shoulder and arm forward against resistance or complete abduction of the arm brings it into action. A subject with paralysis of the serratus magnus cannot raise the arm higher than to a horizontal position, either forward or sideward, even though the trapezius and the deltoid are functioning normally (Figs. 6 and 7). When attempting to do so, the whole vertebral border protrudes. There is no tipping forward of the scapula and the vertebral

border remains parallel to the spinal column. The trapezius provides a certain fixation of the scapula, but it is not able to rotate the scapula upward, hence the arm cannot be raised above a horizontal position. Pushing forward against resistance, as in assuming a prone falling position, further exaggerates the winging of the vertebral border of the scapula.

Observations of a number of cases with isolated paralysis of the serratus magnus indicate that no other muscle can take over its function. The pectoralis major muscle may draw the arm and indirectly the whole shoulder girdle forward, but the vertebral border of the scapula will protrude

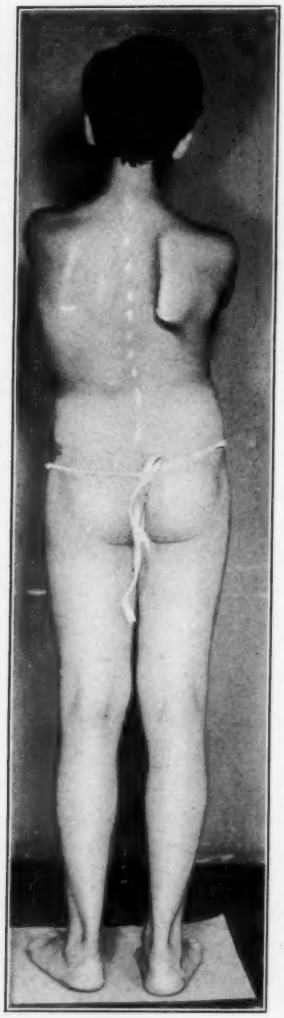

FIG. 6

FIG. 7

unless the serratus magnus with its grip from the inside keeps the scapula flat on the rib cage. The rhomboids may assist in keeping the vertebral border of the scapula close to the rib cage, but these muscles have a downward rotary action on the scapula and, therefore, cannot substitute for the serratus magnus. The trapezius, which seems to have a good anatomical position for bringing about upward rotation of the scapula, does not do so to any considerable extent. The serratus magnus seems to be able to take over the function of the trapezius, but not vice versa. This makes us conclude that the latter muscle is by far the more important of the two.

In subjects lacking both trapezius and serratus magnus, there is no anchorage of the scapula and neither flexion nor abduction of the arm is very successful (Figs. 1 and 8). An attempt to raise the arm sideways brings about abduction of the humerus on the scapula through action of the deltoid, but the scapula rotates downward instead of upward. Since the scapula cannot be held firm, the deltoid pulls the acromion downward, and the weight of the arm also contributes to faulty rotation of the scapula. Complete absence of the upward rotators causes a very serious disability.

FIG. 8

Rhomboids

The rhomboid muscles, when the scapula is in an upward rotated position, pull the whole scapula, and in particular the inferior angle, toward the spinal column. When completing the downward rotary movement they also tend to elevate the shoulder girdle. These muscles are covered by the middle and the lower portions of the trapezius. They can easily be palpated, however, provided the trapezius

FIG. 9

FIG. 10

is relaxed. To secure relaxation of the trapezius the subject should hyperextend the arm at the shoulder and place the hand across the back in the lumbar region. The examiner inserts the index finger or the ulnar border of the hand beneath the scapula (Fig. 9). This meets with no difficulty if the shoulder muscles are relaxed and some counterpressure is given from the front. The patient is then told to raise the forearm and the hand slightly away from the body (Fig. 10). This movement causes the rhomboids to contract powerfully, and the examiner's finger is pushed out from underneath the scapula. In all normal subjects the rhomboids can thus be palpated, but the muscle is most easily observed in subjects lacking the lower trapezius (Fig. 10). If the rhomboids are absent or weak, there is an empty space between the scapula and the rib cage and the fingers are not pushed out when the test is given. A subject with weak rhomboids may be able to raise the hand off the back, but with some difficulty, in which case the pectoralis minor tips the scapula forward. If there is an involvement of the posterior deltoid, the latissimus dorsi, the inward rotators of the arm or the flexors of the forearm, the examiner must support the arm and the forearm when the test is given.

If the rhomboids are weakened or completely paralyzed, and the arm is adducted against resistance, the scapula is pulled toward the arm by the teres muscles and fails to rotate downward. This test for the rhomboids should be given in addition to the test described above, since it presents a very typical picture. However, if the teres muscles are paralyzed as well, this latter test is of no value. Weakness or paralysis of the rhomboids is not noticeable in ordinary elevation and lowering of the arm

as gravity assists the downward rotation of the scapula. It is only when resistance is applied that a weakness becomes evident.

Paralysis of the rhomboids is fairly common in poliomyelitis, but other muscles are very likely to be involved at the same time. Isolated rhomboid paralysis or a congenital absence of the rhomboids has not been observed by the writer of this paper.

Pectoralis Minor

The pectoralis minor, originating from the third. fourth, and fifth ribs and inserting into the coracoid process, is covered by the pectoralis major. By pulling downward on the coracoid process the pectoralis minor tips the scapula forward, causing the inferior angle to protrude. In forced breathing the pectoralis minor comes into action, pulling upward on the ribs, and can be felt contracting, a test suggested by Bowen[1]. However, in this test one cannot prevent the pectoralis major from tightening up as well, which makes palpation of the pectoralis minor quite difficult. To eliminate the contraction of the pectoralis major the subject should assume the same position as that for testing the rhomboids (Fig. 9). The examiner places the finger tips just below and medial to the coracoid process. If the subject is cooperative and able to relax, the examiner can insert the fingers quite deeply and can reach almost underneath the coracoid process without causing the subject any discomfort. When thereafter the subject raises the forearm off the back, the pectoralis minor comes into vigorous contraction and the fingers are pushed out from underneath the coracoid process by the contracting muscle. By palpating the rhomboids and the pectoralis minor simultaneously, it can be determined whether the tipping forward of the scapula is performed by the pectoralis minor alone, or by the rhomboids alone, or whether both muscles participate. Isolated paralysis of the pectoralis minor has not been observed by this writer.

Levator Scapulae

This muscle is most easily palpated in subjects lacking the upper trapezius (Fig. 5). The almost vertical fibers of the levator scapulae, as compared to the more oblique fibers of the upper trapezius, make it possible to distinguish between the two muscles in normal individuals also. When the shoulders are shrugged, the levator scapulae can be felt behind the sternocleidomastoid muscle in the posterior triangle of the neck. A fairly good relaxation of the upper portion of the trapezius is obtained if the subject presses the arm against the side while elevating the shoulder girdle.

The levator seems to be perfectly able to take over the function of the upper trapezius when this muscle is paralyzed. Whether or not the opposite is also true—that is, whether the upper trapezius is able to elevate the shoulder girdle against resistance in case of paralysis of the levator scapulae—cannot be learned from the present study. No cases were observed with this type of paralysis.

SUMMARY

1. A study of seventeen cases of isolated paralysis of the shoulder-blade fixators was undertaken in order to determine the relative importance of each of these muscles in arm movements.

2. Tests for individual muscles are described.

3. Isolated paralysis of the trapezius, even in cases where the whole muscle is absent, does not interfere appreciably with the elevation of the arm. The disability is marked only when the subject abducts the arm with the trunk inclined forward.

4. Isolated paralysis of the serratus magnus causes far more disability than paralysis of the trapezius alone, as the subject is unable to elevate the arm above a horizontal position.

5. Paralysis of both the trapezius and the serratus magnus causes a very serious disability. The scapula completely lacks fixation, and abduction of the arm can be performed to approximately 60 degrees only.

6. Isolated paralysis of the rhomboids or the pectoralis minor was not present in any of the cases studied. The exact effect of the loss of one of these muscles, therefore, could not be determined. In cases with weak rhomboid muscles, resistive adduction of the arm proves to be rather difficult, as the teres major pulls the scapula toward the arm instead of pulling the arm toward the scapula.

7. Isolated paralysis of the levator scapulae was not found in any of the cases studied. When the upper trapezius is absent and the rhomboids weak, the subject has no difficulty in elevating the shoulder girdle against resistance. It seems, therefore, reasonable to assume that in normal subjects, too, the levator scapulae is the most important muscle for elevation of the shoulder girdle.

8. The etiology could not be determined with certainty in all cases, but five groups were recognized: injury to the long thoracic nerve, injury to the spinal accessory nerve, congenital defects, muscle dystrophy, and poliomyelitis.

1. Bowen, W. P.: Applied Anatomy and Kinesiology; The Mechanism of Muscular Movement. Ed. 4. p. 76. Philadelphia, Lea & Febiger, 1928.

EARLY REPAIR OF BONE: AN EXPERIMENTAL STUDY OF CERTAIN FACTORS *

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The textbook description of reparative changes following fracture of bone focuses the attention of the student on the site of the fracture to the exclusion of all other regions. Almost invariably an outpouring of blood and connective-tissue cells from the medullary cavity and cortex is described, together with an associated proliferation of the periosteum and adjacent soft tissues. The blood clot then becomes infiltrated with fibroblasts, which undergo cytological changes from the cartilage stage to formation of adult bone. Certain of these changes do occur, but the earliest and most marked reactions frequently take place not only at the site of fracture, but along the entire shaft as well.

The authors' attention was drawn to this fact in the course of certain experiments which were undertaken to investigate the stimulating effect of embryonic chick extract on repair of fractures. The microscopic appearances noted stimulated them to make a more exhaustive study.

METHOD OF STUDY

Twenty-one mature female rats, each five months of age and weighing about 200 grams, were used. The right humerus of each rat was fractured under anaesthesia at the junction of the middle and lower thirds by the method described in a previous article.[1] Retentive apparatus was not used to immobilize the limb. The rats were arranged in three groups. Group A consisted of seven normal rats in which the humerus was fractured. Group B consisted of seven rats which had been spayed two months previously. Vaginal smears made prior to fracture of the humerus indicated an absence of estrogenic substance in the circulation. The rats of Group C corresponded in all details to those of Group B except that each rat received 2000 international units of estrogenic substance on the second, fourth, and sixth days following fracture.

One rat of each group was killed at daily intervals following fracture. The humeri were removed and preserved in a 10-per-cent. solution of formalin prior to decalcification. Thereafter, the bone was prepared in the usual manner for microscopic examination.

Careful and repeated microscopic examinations of the sections removed from the first to the seventh postoperative day indicated that the

* The work was done at the Institute of Experimental Medicine under the supervision of G. M. Higgins, M.D.

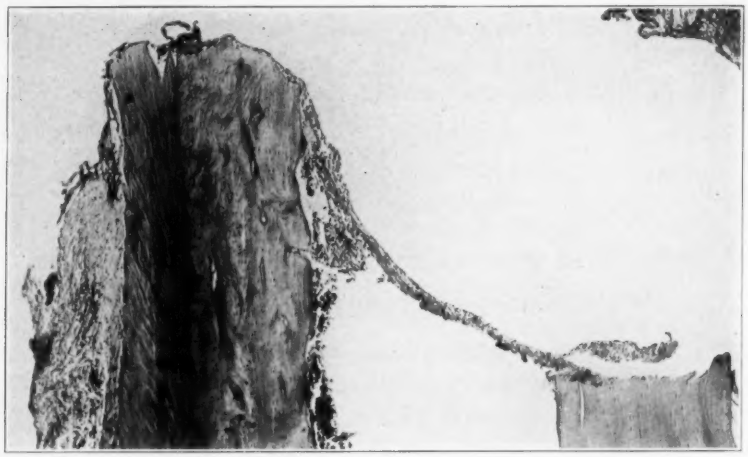

FIG. 1

Photomicrograph (× 50) of tissue obtained on the first postoperative day from a spayed rat which had received estrogenic substance, shows a fibrinous suspension bridge stretched across the medullary cavity from the periosteum on one side to the periosteum on the other.

rate of formation of new bone, especially the endosteal bone, was increased among the rats in Group C which had been given estrogenic substance. In an attempt to demonstrate this formation of new bone satisfactorily a new series of rats, comparable in age and weight to those of Groups A, B, and C, received injections of a 1-per-cent. solution of alizarin prior to and subsequent to operative fracture of the right humerus. Specimens of bone were removed at daily intervals and filed down on the microtome, as this was the only way satisfactory sections could be procured.

In the rats of the last group, a definite zone of formation of new bone, as revealed by the pink coloration of alizarin, appeared four days after fracture. The solubility of the dye makes permanent preparations of sectioned material unsatisfactory, and some other method, therefore, is required to demonstrate grossly the extent of formation of new bone satisfactorily.

MICROSCOPIC FINDINGS

The first specimens, which were examined twenty-four hours after the humeri were fractured, revealed the presence of hemorrhage in the soft tissues. At this early stage a definite capsule uniting the fractured ends of bone was not present. Microscopically, the medullary cavity was isolated by a delicate cellular structure similar to a suspension bridge, which was continuous across the region, joining the periosteum on the two sides (Fig. 1). This structure, when seen under a high-power lens, was found to consist of interlacing strands of fibrinous material containing lymphocytes, leukocytes, and plasma cells (Fig. 2). A fact of some significance, perhaps, was the absence of a corresponding number of red blood corpuscles.

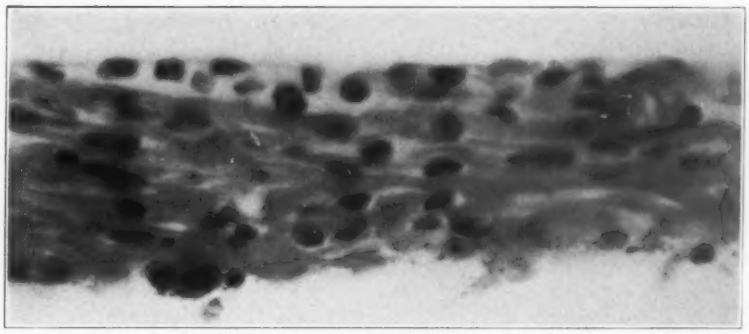

FIG. 2

Photomicrograph (× 600) of tissue obtained on the first postoperative day from a spayed rat which had received estrogenic substance, shows lymphocytes and plasma cells enmeshed in the middle of the fibrinous bridge.

In all probability the bridgelike cellular structure was formed from fibrin, but its appearance suggested that it was composed of matrix, in which red blood cells could develop. In other sections strands of periosteum extended across the medullary cavity, thereby acting as a seal. Along the sides of the shaft the periosteum was normal and firmly adherent to the underlying bone.

The gross appearance of the specimens removed two days after the bone was fractured was similar to that just described, but in addition the soft tissues immediately adjacent to the fractured ends had a whitish, rather glistening appearance, were attached to the ends of the bone, and seemed to act as a capsule. This tissue was extremely delicate and could be torn easily.

Microscopic examination of tissue removed on the second postoperative day demonstrated cytological features at the site of the fracture, similar to those found twenty-four hours after fracture (Fig. 3). The most pronounced change was the increased thickness of the periosteum of the entire bone, which was as marked near the surgical neck as it was at the site of the fracture. It appeared as though some circulating stimulant had resulted in general periosteal hyperplasia.

Sections of bone removed from the spayed rat indicated that a small fragment of the cortex had become detached and acted as a plug in the medullary cavity (Fig. 4), sealing it effectively. The question arose as to whether fusion between this hard cortical plug and the adjacent cortex could lead to subsequent non-union.

Three days after fracture there was little change in the gross appearance of the specimens from that found on the second postoperative day. Microscopically, however, the fibrinlike seal covering the medulla was thicker, and apparently invaded by fibrous-tissue cells which had extended into the marrow. These sections demonstrated well the continuity of this organized plug with the surrounding periosteum (Fig. 5). The periosteum was markedly thickened, and at positions well removed

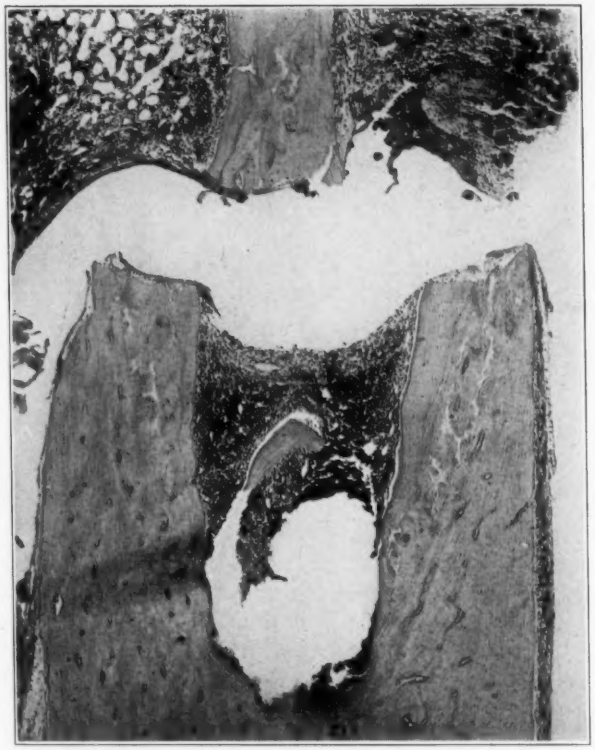

FIG. 3

Photomicrograph (× 42) of tissue obtained on the second postoperative day from a rat of Group A (normal except for the fracture), shows sealing of the medullae of both fragments by a cordon of fibrin continuing in the periosteum across both ends of the fractured bone.

FIG. 4

Photomicrograph (× 25) of tissue obtained on the second postoperative day from a spayed rat, shows a plug of cortical bone in the medullary cavity. Could this be a potential cause of non-union? The periosteum is thickened.

FIG. 5

Photomicrograph (× 35) of tissue obtained on the third postoperative day from a rat which was normal except for fracture, shows the sealing of the medulla, the covering of which now appears to be organized, to be more marked than in Fig. 3. Its continuity with the much thickened periosteum, and early osteoid formation under the periosteum, and a trace of beginning osteoid formation in the endosteum can also be seen.

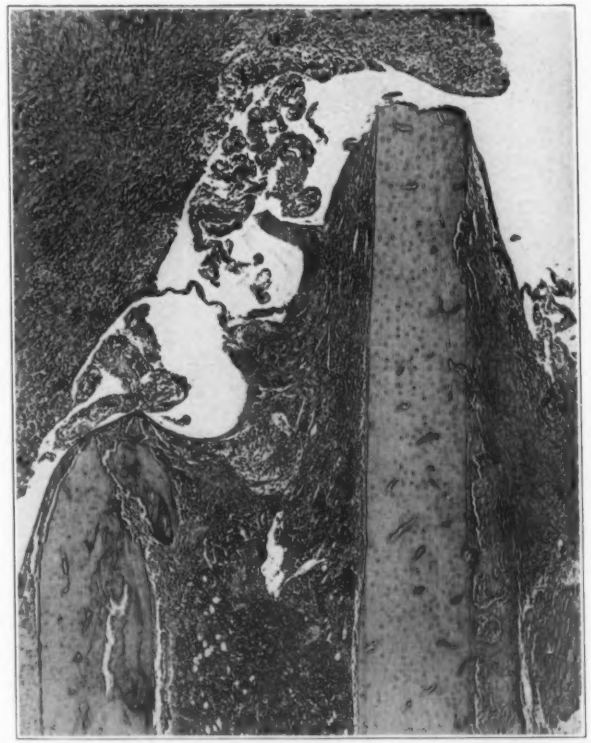

FIG. 6

Photomicrograph (× 30) of tissue obtained on the sixth postoperative day from a rat which was normal except for fracture, shows a marked degree of subperiosteal formation of new osteoid tissue just below the surgical neck. Formation of new bone within the region well removed from the site of the fracture can be seen.

from the site of injury it revealed the presence of young osteoid tissue (Fig. 6). At the site of the fracture the soft tissues had been infiltrated by round cells and leukocytes; young blood vessels had made their appearance in the marrow and it seemed that a concentration or perhaps proliferation of megakaryocytes in these regions had also taken place. Young osteoid tissue had developed from the endosteum and stretched down toward the extremity of the bone.

The sections prepared from the specimens removed four days following fracture of the humerus were similar to those described previously, but in a slightly more advanced stage of development. In addition, a few osteoclasts were seen at the ends of the bone. Osteoid tissue was not present between or in the soft tissues adjacent to the fractured ends.

By the fifth day a fairly well-developed capsule united the ends of the bone. This capsule had a whitish, glistening appearance, and was much more tenacious than that described in the specimen removed two days after fracture. Subperiosteal osteoid tissue developed at sites well removed from the fracture and small islands of young cartilage appeared, but they were secondary in size and date of origin to the osteoid tissue.

Well-developed endosteal bone rapidly sealed the medullary cavity, and in relation to it young blood vessels were seen in the marrow. Osteoclasts or fused bone cells liberated from the fragmented ends of bone also were present.

The specimens removed on the sixth postoperative day revealed further development of the features just described (Fig. 6). Subperiosteal and endosteal formation of new bone was marked, and in the former the development of cartilage was recognized. A more definite sealing of the medulla was noted and in the surrounding tissues a reversal to a more embryonic type of cell was apparent.

By the seventh postoperative day the ends of bone were united by a comparatively tough capsule composed of closely packed and elongated fibroblasts. In some instances this capsule was bilocular, as it was divided by a central septum into two cavities, each one adjacent to the fractured end of one of the fragments of bone but separated from its medullary cavity by the endosteal plug. The cavities contained a network of interlacing fibers, the interstices of which were filled with amorphous débris. The walls in certain regions disclosed the presence of myxomatouslike cells. When the walls were cut, a sanguineous fluid exuded from the cavities. It is possible that within this capsule nature has developed a small chemical factory in which the hydrogen ion is controlled and bone is developed.

The development of subperiosteal and endosteal osteoid was well demonstrated in all the sections made on the seventh postoperative day.

COMMENT AND CONCLUSIONS

Sealing of the medullary cavity appears to be one of the first steps in the healing of a fracture. This may be produced by (1) a strand of peri-

osteum, (2) a band of fibrin, or (3) a fibrinouslike plug produced by mass destruction of the red corpuscles. The seal is continuous across the cortex with the periosteum, and later becomes infiltrated by fibrous-tissue cells from this membrane. In other cases, however, these cells may arise from the marrow or develop directly from the lymphocytes caught in meshes of this fibrinous medullary cap.

By the second day, the periosteum from the site of fracture to the neck of the humerus is thickened. This would appear to be the result of a general stimulation rather than that caused by local trauma. By the third day, osteoid tissue has developed subperiosteally in regions well removed from the fracture. At the same time, osteoid tissue which has arisen from the endosteum, has also made its appearance at the fractured ends of the bone and, in conjunction with the original fibrinous plug, formed a more efficient seal for the medullary cavity. The development of this subperiosteal bone along the entire shaft was one of the most interesting features of this study.

The authors were not able to demonstrate any appreciable differences in the early stages of repair of bone in the three groups, but gained the impression that injections of estrogenic substance stimulated production of endosteal osteoid tissue. The insertion into the medullary cavity of a small plug of dense cortical bone as occurred in one of the cases could predispose to non-union.

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LONGITUDINAL FISSURES IN THE VERTEBRAL ARTICULAR PROCESSES

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Radiolucent gaps in the neural arch and its processes are known to be due to persistence of cartilaginous union and failure of bony union between parts which arise from distinct centers of ossification. Most of these so-called fissures are in the laminae, in the isthmus between the superior and inferior articular processes, and at the tip of the inferior articular processes of the lumbar vertebrae. An additional fissure, apparently not yet recorded, was found in four persons. In each of them, it was associated with gaps at the tips of the articular process and, in one patient, with gaps in both the isthmus and the lamina of the same vertebra. It extended from the region of the isthmus along the longitudinal axis of the articular process down to the gap at its tip, the

FIG. 1-A

FIG. 1-B FIG. 1-C

Anteroposterior (Fig. 1-A) and oblique (Fig. 1-B) views of the mid-lumbar region of a man, aged thirty-two, examined for hematuria. The right third inferior lumbar articular process has a gap at its tip and a longitudinal fissure with irregular outlines. The two gaps are retraced for better demonstration in Fig. 1-C. There are no symptoms referable to the spine, or any history of trauma.

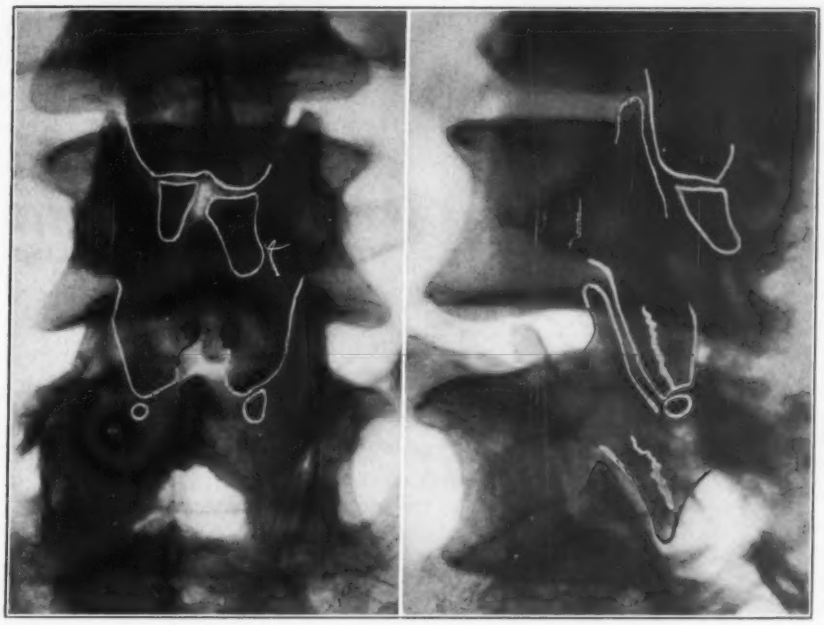

FIG. 2-A FIG. 2-B

Anteroposterior (Fig. 2-A) and oblique (Fig. 2-B) views of the lower lumbar region of a man, aged sixty-four, examined for a tumor in the epigastrium. There were no signs or symptoms of vertebral disease, and no history of trauma. Note gap formations in the inferior articular processes of the third and fourth lumbar vertebrae, with longitudinal fissures (outlines retraced).

two fissures standing at right angles (Figs. 1-A, 1-B, and 1-C). In all the cases the outlines of the longitudinal fissure were irregularly serrated, which suggested a fracture line. None of the patients, however, remembered having incurred an injury, and there were no signs or symptoms whatever referable to the spine. The gaps were discovered accidentally in roentgenograms of the digestive or urinary tract. Since there is only one center of ossification for the main part of each articular process, according to present knowledge, it would seem difficult to account for the longitudinal gap by a developmental failure of bony union. However, the constant combination with a second gap known to be due to such a failure, together with the absence of a history or clinical signs of trauma, is in favor of a developmental anomaly. These longitudinal fissures are rare, their incidence being in our observations as low as that of aplasia of articular processes,—namely, 0.09 per cent. of all spines examined.

The sole significance of the longitudinal gaps would seem to lie in the fact that they look like fracture lines. Fractures of the articular processes are rare, most of them being combined with fractures of other parts of the vertebrae. The writer has observed only three cases of fracture of an articular process without other vertebral lesions; in each of them, the clinical signs and symptoms were definite and the trauma had been severe. Two of them had radicular neuritis with atrophy of the corresponding

muscles and bone, and in all the cases the mobility of the spinal section involved was greatly diminished. The fact that a longitudinal fissure resembling a fracture line may exist in the complete absence of clinical manifestations may be worth recording, since increasing importance is given to roentgenographic findings in deciding on questions of treatment and of compensation after some relatively slight injury. One may easily be inclined to ascribe symptoms to such a formation, when they are perhaps due to some other cause,—for instance, to a simple strain or a post-traumatic neurosis.

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CALCIFICATION AND OSSIFICATION

II. Control of Calcification in the Fracture Callus in Rachitic Rats *

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In the preceding paper [20] we have described the progress of calcification in the fracture callus of normal rats. For the purpose of analyzing the process of calcification, observations upon normal animals have definite limitations. Calcification occurs simultaneously with osteogenesis,‡ and there is no possibility of controlling it, or of observing the processes of osteogenesis and of calcification separately. To a very considerable degree these difficulties are avoided in the case of low-phosphate rickets. If this form of rickets may be regarded as primarily a phosphate deficiency [13] the results obtained may be safely transferred to the interpretation of processes occurring under more normal conditions. Calcification and healing can be induced in low-phosphate rickets in the rat by the administration of phosphates; and the healing so obtained is comparable with that produced by the administration of vitamin D [12].

I. HEALING FRACTURES IN UNTREATED FLORID RICKETS

Experimental studies concerned with the relationship of diet to the healing of fractures have been reviewed by Goisman and Compere. Erdheim; Downs and McKeown; Ham, Tisdall, and Drake; and Compere, Hamilton, and Dewar have described decalcified histological preparations of healing fractures in rachitic animals. Pappenheimer observed fractures in rachitic rats, in sections stained by the von Kóssa method after partial decalcification in Müller's fluid.

Rats were weaned to the Steenbock-Black rachitogenic diet No. 2965 at the age of twenty-one days and were given fractures at the age of seven weeks. The animals were sacrificed at daily intervals from one to fifteen days following the fractures and at two-day intervals thereafter. The series of preparations includes the bones of five litters of rats with florid rickets and the fractures in untreated rachitic rats used as controls in

* This work was aided by a grant from the Josiah Macy, Jr., Foundation.

† Henry Strong Denison Scholar for 1940–1941.

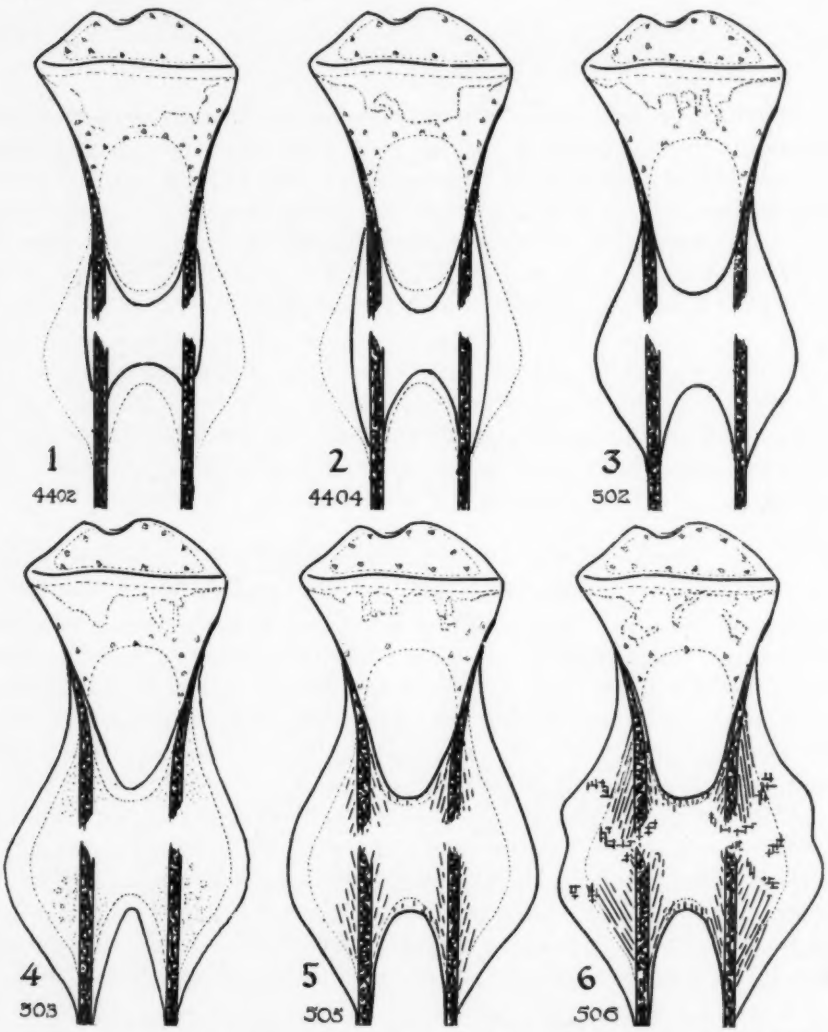
‡ Properly speaking the terms osteogenesis and ossification include the calcification which is an essential part of the formation of bone. In order to avoid confusing circumlocutions we have at times separated these meanings, so that osteogenesis and ossification are used to designate the formation of osseous tissue, without regard to its state of calcification. Similarly we have used the terms osteocyte, osteoblast, and osteoclast to designate particular types of cells, without reference to whether the osseous tissue with which they are associated is calcified or not.

twenty other experiments. The techniques employed in this study were those previously described [20].

In rachitic rats the healing of fractures begins as it does in normal rats [20] with the important difference that the healing process proceeds in

FIGS. 1–18

Figures 1–18 are diagrammatic representations of deposits of bone salt in rachitic bones with healing fractures.†

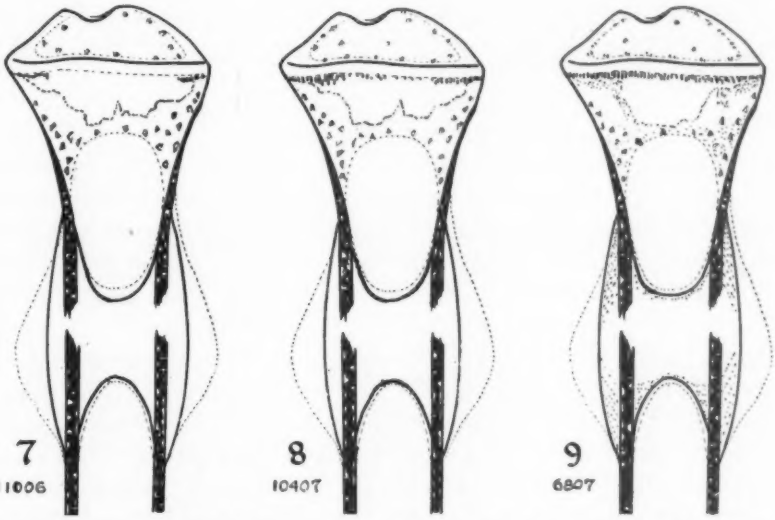


Healing fractures in untreated rickets. Fig. 1: after two days; Fig. 2: four days; Fig. 3: eight days; Fig. 4: twelve days (note beginning spontaneous calcification in subperiosteal and subendosteal osteoid); Fig. 5: twenty days; and Fig. 6: non-union at twenty-eight days, with considerable spontaneous calcification in the callus and none in the epiphyseal cartilage or metaphysis.*

† For similar diagrams of healing fractures in normal rats, see Urist and McLean [20].
* See key on page 285.

the complete absence of calcification of the newly formed tissues for the first ten to fifteen days following the injury (Figs. 1, 2, 3, 20, 27, and 31). There is the same initial inflammatory response to the injury, resulting in the formation of the fibrocartilaginous callus in and around the fracture line. There is the same formation of osseous tissue under the periosteum and the endosteum, beginning at some distance from the fracture line, elevating these membranes, and advancing toward the callus mass. In the early stages of healing the osseous tissue is indistinguishable from similar tissue formed in the animal on normal diet, unless stained to detect the absence of calcification.

The differences in the reaction of the rachitic animal to a fracture from those of a normal animal with a similar injury, exclusive of the state of calcification, begin to be apparent by about the fourth to the fifth day following the fracture. At this time, in the normal animal, the new intramembranous bone, formed under the periosteum and the endosteum, has



Healing fractures in rickets four days after fracture following a single dose of one-tenth molar of phosphate solution, 2.5 cubic centimeters per 100 grams body weight. Fig. 7: four hours after administration of phosphate; Fig. 8: twelve hours; Fig. 9: twenty-four hours. Note increasing calcification in epiphyseal cartilage and beginning calcification in subperiosteal and subendosteal osteoid.*

Normal shaft

Beginning calcification

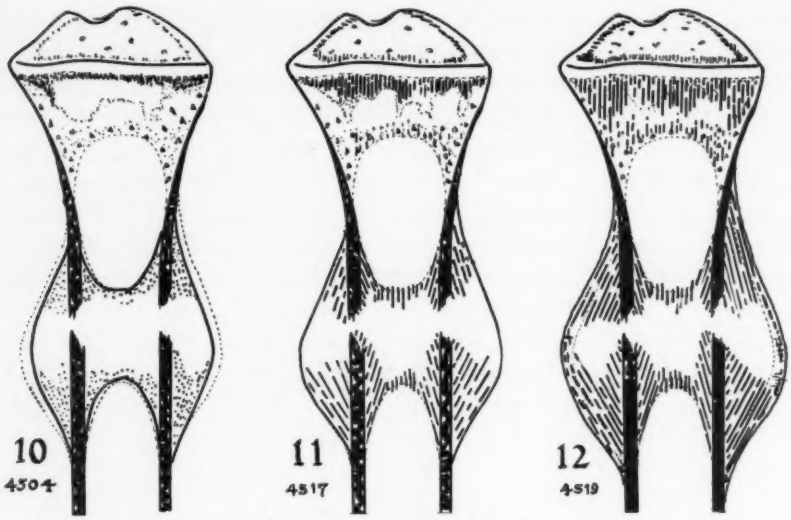
Osteoporotic shaft

Calcified cartilage

Spongy bone

Old calcified trabeculae with osteoid borders.

Light broken lines represent the usual limits of epiphyseal bone, epiphyseal cartilage, rachitic cartilage, and chondro-osteoid, spongiosa, and fracture callus, respectively.



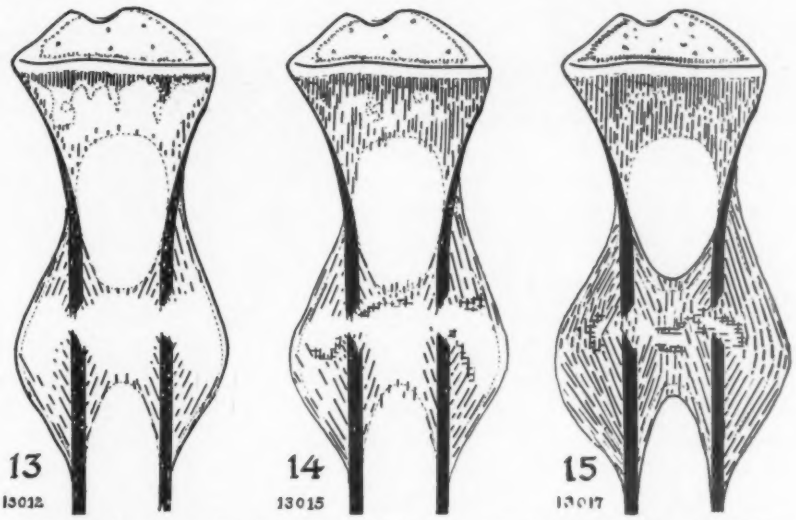
Healing fractures after daily doses of phosphate, beginning with the fourth day of healing. Fig. 10: after five days of healing; Fig. 11: eight days; Fig. 12: twelve days. Note increasing calcification in callus, metaphysis, and epiphyseal cartilage.*

gained contact with the fibrocartilaginous callus, and removal of the latter with its replacement by bone is well under way. The fibrocartilaginous callus consists at this time of undifferentiated dense connective tissue, of fibrocartilage, and of hyaline cartilage in various stages of development. All of these tissues, in the normal animal, are invaded by the new osseous tissue with equal ease, and their removal proceeds over a wide front with some differences in detail according to the tissue being invaded. At the same time, in the rachitic animal, the callus consists of the same tissues, and is microscopically indistinguishable from the callus in the normal animal. There is, however, in the rachitic animal, a marked lag in the processes of replacement of the callus tissues by the newly formed osseous tissue, and the details of this difference in behavior, and of its correction when treatment leading to deposition of bone salt is instituted, indicate the degree to which the processes in the normal animal are dependent upon the orderly progress of calcification.

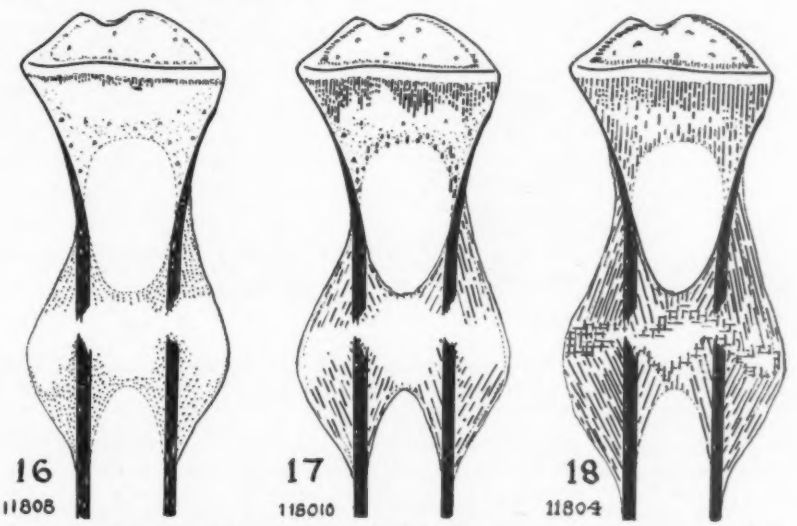
Park has described the process of cartilage removal in the rachitic rat as an invasion of the cartilage by capillary "bushes", leading to its replacement by uncalcified "pseudo-osteoid". In rachitic animals, as in normal animals, there are invasion and replacement of the epiphyseal cartilage plates. The essential difference in rickets is that, in addition to the failure of the cartilage matrix to calcify, invasion by the capillary "buds" or bushes occurs over a front of several rows of cartilage cells together, instead of invasion of the rows singly. Also, instead of only cartilage matrix, stripped of its cells, being left, there are columns of cartilage, of varying widths and thicknesses, in which cartilage cells persist.

* See key on page 285.

As in normal intracartilaginous ossification, there is apposition of osseous tissue upon a framework of tissue derived from the epiphyseal cartilage. In rickets this tissue is uncalcified osteoid, and it is laid down upon the persisting remnants of cartilage which, as stated above, include cartilage cells. The result is that the rachitic metaphysis is made up of uncalcified trabeculae, which include cells with the arrangement and some of the



Healing fractures after daily doses of phosphate, beginning with the eighth day of healing. Fig. 13: after ten days of healing; Fig. 14: sixteen days; Fig. 15: partial union at twenty-four days.*



Healing fractures after the forced administration of the maximum amount of phosphate tolerated, beginning on the eighth day of healing. Fig. 16: after ten days of healing; Fig. 17: eleven and one-half days; Fig. 18: twelve and three-quarters days. Note rapid increase in calcification.*

* See key on page 285.

FIG. 19

Photomicrograph (× 8) of longitudinal section through undecalcified tibia, ten days after fracture, of a rat, fifty-nine days of age, which was weaned at twenty-one days to Steenbock-Black rachitogenic diet No. 2965 supplemented by 2-per-cent. cod-liver oil. The epiphyseal cartilage appears somewhat wider than normal. There is delay in calcification in the epiphyseal cartilage, metaphysis, and subperiosteal new bone. Silver nitrate-hematoxylin-eosin stain was used in this and the following specimens.

morphological characteristics of the epiphyseal cartilage cells, and which are encased in osteoid. The fate of these cartilage remnants has been considered in detail by Dodds and Cameron and need not

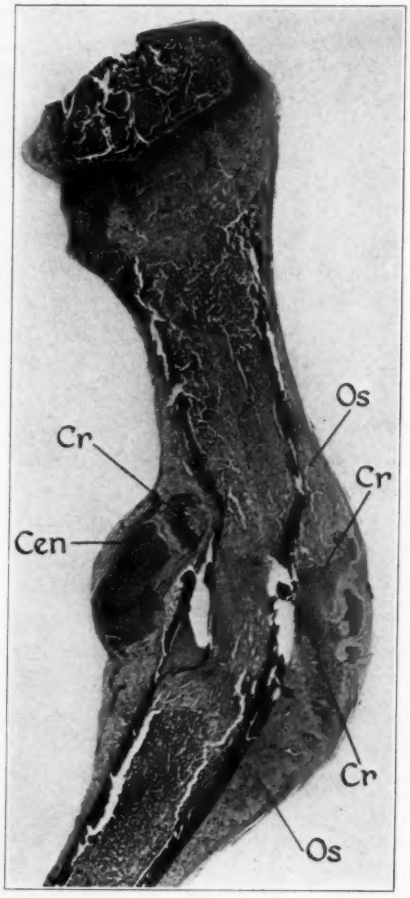


FIG. 20

Photomicrograph (× 8) of longitudinal section through undecalcified tibia, ten days after fracture, of a rat, fifty-nine days of age, which was weaned at twenty-one days to Steenbock-Black rachitogenic diet No. 2965. There is complete absence of calcification in the epiphyseal cartilage, metaphysis, and callus. *Cr*, cartilage in process of removal, rachitic type; *Cen*, partially encapsulated cartilage, not being removed; *Os*, subperiosteal osteoid, in contact with callus cartilage.

be described here. The rachitic metaphysis as a whole is a mixture of uncalcified tissues derived from cartilage and from the osteogenic cells of the advancing bone marrow. This tissue was called "chondro-osteoid" by von Recklinghausen and we shall use the term for this atypical tissue arising in both the metaphysis and the callus.

The process, just described, of intracartilaginous ossification from the epiphyseal cartilage, is duplicated in the callus cartilage in rachitic animals (Fig. 31). But, just as there is a delay of some days [4] in the initiation of this process in the epiphyseal cartilage following the onset of rickets, so there is a lag in its initiation in the callus of the rachitic animal. Under the most favorable conditions for its initiation and progress, which are that the new intramembranous osseous tissue is in contact with the hypertrophic cartilage, the growth of buds or bushes into the callus cartilage does not begin until about the ninth or twelfth day after the fracture. From this time on the process may progress rapidly, eventually leading to complete replacement of the callus cartilage with chondro-osteoid, and to actual union across the fracture line. The picture, however, is complicated by the occurrence of spontaneous calcification as described below.

In many instances, there is an additional factor which further complicates and delays the process of replacement of the callus by osseous tissue. It has been stated above that in normal animals the new osseous tissue invades undifferentiated fibrous tissue, fibrocartilage, and hyaline cartilage with equal ease. Consequently when fibrous tissue or fibrocartilage lies between the advancing bone and the hyaline cartilage of the callus, this intervening tissue is early replaced by bone, and offers no barrier to the advance of the osseous tissue. In the rachitic animal, however, invasion of connective tissue or of fibrocartilage, if it occurs at all, is accomplished slowly and after considerable delay. During the period of delay incident to the invasion of the callus cartilage, the latter frequently becomes embedded—one might almost say encapsulated—in dense connective tissue and fibrocartilage (Fig. 20). This encapsulation not only forms a barrier to the replacement of the callus cartilage by osteoid tissue, but it also forms a barrier to the resumption of the normal healing process when calcification of the callus is instituted by suitable means. This latter influence of the encapsulation of the callus cartilage is considered in detail in the following sections.

Spontaneous Calcification in the Callus

The processes just described, in the absence of calcification, tend toward the union of the fracture by the formation of a callus composed of osteoid tissue. Before this point is reached, however, the process is modified by the deposition of bone salt in the callus.

As stated above, the callus in florid rickets is characterized by a complete absence of bone salt during the first ten to fifteen days of its development. At about this time, however, there appeared regularly in our experiments (Figs. 4, 5, and 6) a beginning calcification in the osteoid of

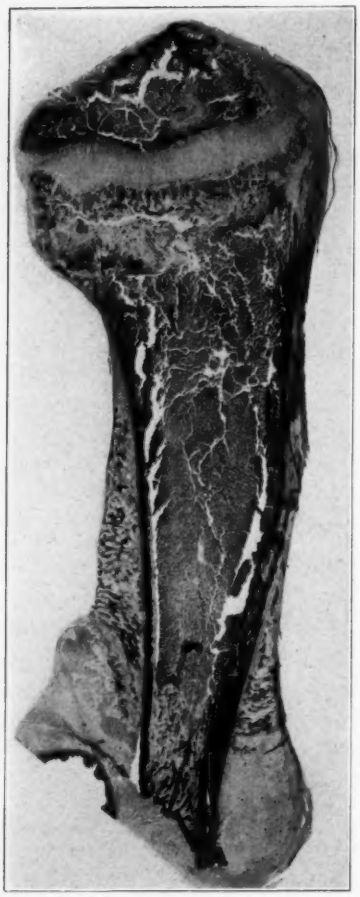


FIG. 21

Photomicrograph (× 8) of longitudinal section through the undecalcified tibia, twelve and one-half days after fracture, of a rachitic rat which had been injected daily with 2.5 cubic centimeters of one-tenth molar of phosphate mixture per 100 grams body weight from the tenth to the twelfth day of healing. There is early calcification and healing in the epiphyseal cartilage, rachitic metaphysis, and callus.

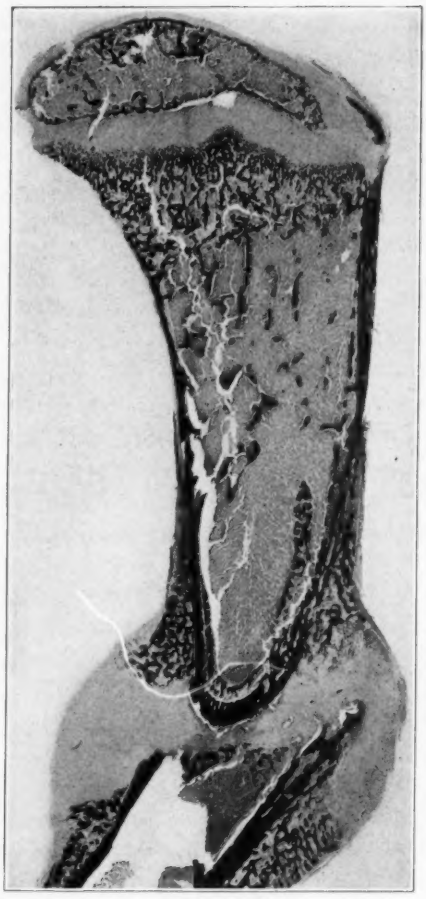


FIG. 22

Photomicrograph (× 8) of longitudinal section through the undecalcified tibia, twelve days after fracture, of a rachitic rat which had been injected daily with 2.5 cubic centimeters of one-tenth molar phosphate mixture per 100 grams of body weight from the fourth to the twelfth day of healing. There is advanced calcification and healing in the epiphyseal cartilage, metaphysis, and callus.

the callus, without a corresponding process in the epiphyseal cartilage or rachitic metaphysis. In order to distinguish this calcification from that induced by the various procedures to be described later we have designated it as *spontaneous calcification.*

Spontaneous calcification appears first in the subperiosteal and subendosteal osteoid at the tenth to the fifteenth day following the fracture. It continues thereafter at a very slow rate. In the hypertrophic cartilage of the callus the time of appearance has been less regular, usually occurring

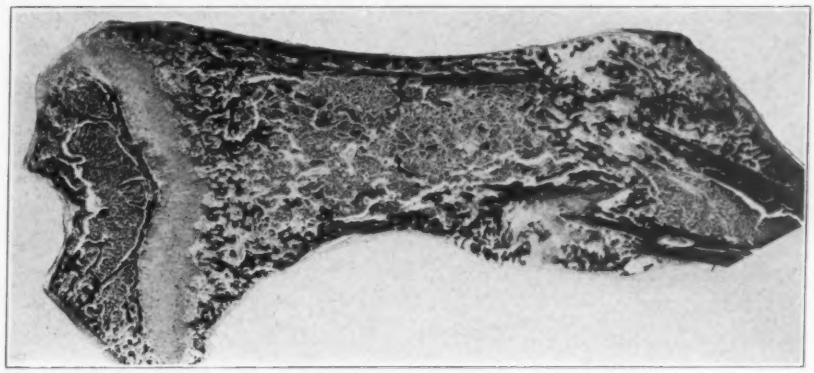


FIG. 23

Photomicrograph (× 8) of longitudinal section through the undecalcified tibia, twenty-two and one-half days after fracture, of a rachitic rat which was injected daily with 2.5 cubic centimeters of one-tenth molar of phosphate mixture per 100 grams of body weight from the fifteenth to the twenty-first day. There is heavy calcification and beginning of union in the fracture callus. Interruption of the healing of rickets is revealed by failure of calcification and resumption of the rachitic type of cartilage removal in the epiphyseal cartilage.

from about the twentieth to the twenty-fifth day, by which time the subperiosteal callus shows a widely distributed deposit. As in the case of induced calcification, described in the following pages, progress of calcification into the callus cartilage is delayed by the encapsulation of the callus cartilage by dense connective tissue and fibrocartilage.

The mechanism of this spontaneous calcification will be considered further in a later paper. This form of calcification is not capable of converting the callus osteoid into fully calcified bone (Figs. 4, 5, and 6). The amount of bone salt deposited is so slight, and the process is so gradual, that, in ordinary roentgenograms, as shown in the illustrations of the papers of Ham, Tisdall, and Drake; and of Compere, Hamilton, and Dewar there is no evidence of calcification throughout the first twenty-five days of healing. In time the callus diminishes in size, is reorganized, and usually, about ten to twenty days after union of the fracture would occur in normal animals, the density of the calcium salts increases sufficiently to become radio-opaque.

II. INITIATION OF CALCIFICATION IN THE CALLUS BY ADMINISTRATION OF A SINGLE DOSE OF PHOSPHATE

Initiation of calcification in the callus was best demonstrated in rats fifty-four days old, five days after a fracture and twenty-four hours following the administration of a single standard dose of a standard phosphate solution.

Rats, weaned to the rachitogenic diet at the age of twenty-one days and given fractures at the age of forty-nine days, were given phosphate solution intraperitoneally four days after the fracture and were killed twenty-four hours later. The standard phosphate solution used for this

purpose was a mixture of 80 per cent. of one-tenth molar secondary sodium phosphate (M/10 Na_2HPO_4) and 20 per cent. of primary sodium phosphate (M/10 NaH_2PO_4), of which the hydrogen-ion concentration at 38 degrees centigrade corresponded approximately to pH 7.35. The standard dosage employed was 2.5 cubic centimeters (containing approximately 7.5 milligrams of phosphorus) per 100 grams of rat weight. This dose is subtoxic for rachitic rats, rarely resulting in the death of an animal in tetany. Increasing the dosage did not shorten the time required for initiation of calcification in such animals as survived the larger dose (Group R, Table II).

Calcification

As previously shown [14] administration of the standard dose of phosphate results in a zone of calcification in the matrix of the epiphyseal cartilage, typical of the "line test", within twenty-four hours or less. In animals examined from twenty-four to forty-eight hours after administration of phosphate and from five to six days following a fracture there was also beginning calcification in the callus, limited to the subperiosteal and subendosteal osteoid. In preparations fixed with neutral formalin and stained with silver nitrate, the initial deposit was seen as widely dispersed rosette-shaped crystals (Fig. 25). In preparations frozen and dried by the Altmann-Gersh method [8], the deposit was in the form of fine non-crystalline granules (Fig. 24). The distribution of the deposits in the callus was the same after each method of fixation.

Silver staining of the early deposit of bone salt cannot be said to demonstrate the exact stage of aggregation of the salt, but undoubtedly it does demonstrate the density and distribution of this mineral. We have compared the localization of the bone salt, as demonstrated by the silver method described, with that shown by *intra vitam* staining by sodium alizarin sulfonate, by observation of unstained sections with the aid of polarized light, and with the fluorescence seen in calcified areas in ultraviolet light. In all cases the distribution of the bone salt was seen to be identical.

Initiation of Calcification at Various Stages of Healing

Table I illustrates the results of a series of experiments designed to investigate the relative receptivity, at various stages of fracture healing and at various intervals during the period on deficient diet, of the growing osteoid and cartilage to bone salt, in both the rachitic metaphysis and the callus. The rats were given a single injection of phosphate as described above and were killed at intervals up to twenty-four hours following the administration of the phosphate.

Table I shows that after the callus had developed a calcifiable matrix, at three to four days following a fracture (Figs. 7, 8, and 9), the initial deposit of bone salt was seen in the subperiosteal osteoid some four to eight hours later than in the epiphyseal cartilage (Fig. 26). After ten

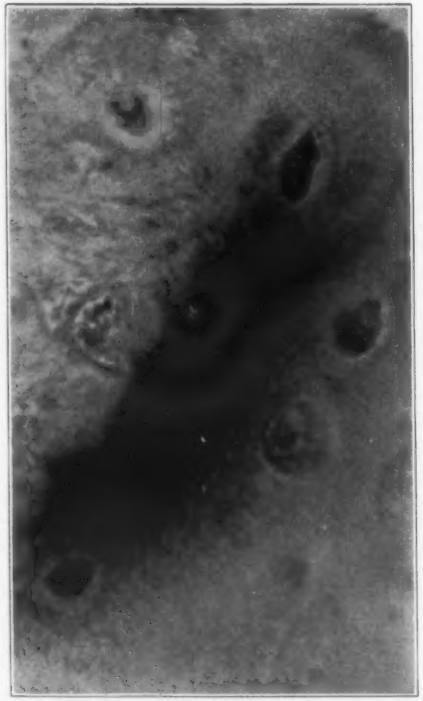


FIG. 24

Photomicrograph (× 850) shows initial calcification in the callus osteoid following the administration of the phosphate mixture. The specimen was frozen-dried, undecalcified, impregnated with 40-per cent. silver nitrate, and exposed to light.

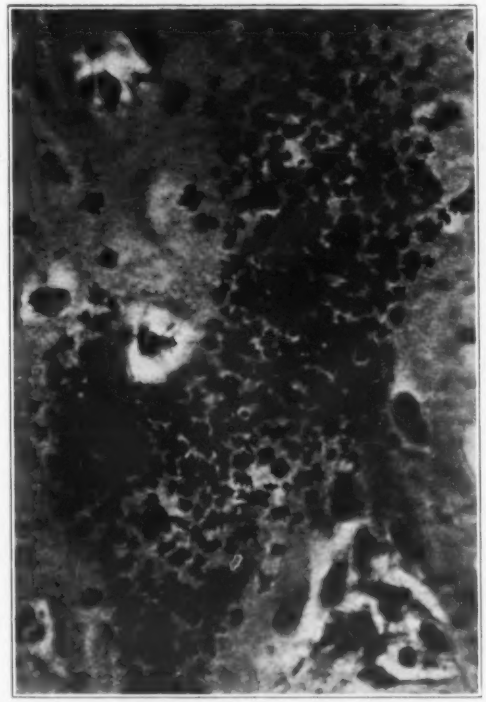


FIG. 25

Photomicrograph (× 800) shows initial calcification in the callus osteoid following the administration of the phosphate mixture. The undecalcified specimen was fixed in neutral formalin.

FIG. 26

Photomicrograph (× 80) shows progressive calcification of the callus osteoid beginning in the area in contact with the periosteum and moving inward ten days after fracture in rachitic rat and forty-eight hours after the first of two daily injections of the phosphate mixture.

TABLE I

INITIATION OF CALCIFICATION IN THE EPIPHYSEAL CARTILAGE, METAPHYSIS, CALLUS OSTEOID, AND CALLUS CARTILAGE IN RACHITIC RATS, FOLLOWING INTRAPERITONEAL ADMINISTRATION OF A SINGLE DOSE OF M/10 PHOSPHATE MIXTURE, 2.5 CUBIC CENTIMETERS (7.5 MILLIGRAMS PHOSPHORUS) PER 100 GRAMS OF BODY WEIGHT

GROUP	Number of Days of Healing	Total Number of Days on Diet	Number of Hours after Injection	NEW CALCIFICATION			
				Epiphyseal Cartilage	Metaphysis	Callus Osteoid	Callus Cartilage
A	0	28	0	−	−		
	0		8	−	−		
	0		24	+	−		
	0		24	+	−		
B	3	31	0	−	−	−	−
	3		4	−	−	−	−
	3		8	−	−	−	−
	3		12	+	−	−	−
	3		24	+	+	+	−
C	4	32	0	−	−	−	−
	4		4	−	−	−	−
	4		8	−	−	−	−
	4		10	−	−	−	−
	4		12	−	−	−	−
	4		14	+	−	−	−
	4		24	+	+	+	−
D	5	33	0	−	−	−	−
	5		1	−	−	−	−
	5		2	−	−	−	−
	5		3	−	−	−	−
	5		4	+	−	−	−
	5		5	+	−	−	−
	5		6	+	+	−	−
	5		8	+	+	+	−
E	7	35	0	−	−	−	−
	7		14	−	−	−	−
	7		24	+	±	+	−
F	8	36	0	−	−	−	−
	8		0	−	−	−	−
	8		0	−	−	−	−
	8		24	+	±	+	−
G	10	38	0	−	−	+	−
	10		0	−	−	+	−
	10		0	−	−	+	−
	10		4	−	−	+	−
	10		12	−	−	+	−
	10		24	−	−	+	−
	12	40	4	−	−	+	−
	12		8	−	−	+	−
	12		24	−	−	+	−
H	12	41	0	−	−	+	−
	15	43	0	−	−	++	−
	15		0	−	−	++	−
I	16	44	0	−	−	++	−
	20	48	0	−	−	++	±
	26	54	0	−	−	+++	+
	28	56	0	−	−	+++	++

days following a fracture and thirty-eight days on the rachitogenic diet it was more difficult to initiate calcification in the epiphyseal cartilage (Fig. 27). Later, frequently after fifteen days, there appeared spontaneous calcification in the callus osteoid. The density of the deposit in the callus

osteoid increased up to twenty-eight days following the fracture, and in animals examined twenty-six and twenty-eight days after a fracture there was also spontaneous calcification in the matrix of the callus cartilage.

In one litter of rats (Group D, Table I) the bones were studied by means of unfixed, hand-cut, quick-stained (10-per-cent. silver nitrate) sections. In these preparations calcification was observed as reported by McLean and McCoy, using the same method. Calcification in the epiphyseal cartilage was seen at four hours, in the osteoid of the metaphysis at six hours, and in the callus osteoid at eight hours.

III. CONTROL OF CALCIFICATION BY CONTINUED ADMINISTRATION OF PHOSPHATE

In the first series of experiments to be reported in this section, daily injections of phosphate were begun either before or at the time of fracturing, or on the fourth day following the fracture, and continued for several days. As the state of the rachitic epiphyseal cartilage and of the rachitic metaphysis was almost identical in all of the animals, and as the response to the administration of phosphate was uniform this response may be described first.

Within twenty-four hours after the injection, calcification was initiated in the epiphyseal cartilage, in the form of the "line test", as described above. Within forty-eight hours, and following two injections of phosphate, calcification was usually observed in the osteoid zone in the metaphysis, and in the osteoid borders on the old trabeculae of spongy bone at the junction of the rachitic metaphysis with the shaft. With further doses deposition of bone salt continued from both directions toward the central portion of the metaphysis, mainly chondro-osteoid, leaving this portion as the last site to be calcified or removed in the process of regeneration of the spongiosa (Figs. 7, 8, 9, 10, 11, and 12).

Shortly after calcification begins in the cartilage, orderly penetration and removal of cartilage by advancing capillaries begins. These phenomena constitute the beginning of *healing* of rickets, which follows and depends upon calcification, but which is recognizable as distinct from the calcification process itself. A definite time interval elapses between calcification and the cellular evidences of healing, so that calcification may be observed without healing, but as will be reported elsewhere, we have never observed the cellular evidences of healing without calcification.

Injections of Phosphate from the Fourth to Ninth Day and from the Fourth to Twelfth Day Following Fractures

As described above, calcification in the subperiosteal and subendosteal callus osteoid was initiated within twenty-four hours after a single dose of phosphate. Daily injections were continued and the animals were sacrificed at daily intervals to the ninth day. The deposits of bone salt showed a gradual increase in density and in area of distribution (Figs. 10, 11, and 12). After the second injection, many of the dispersed de-

posits became denser and new patches of beginning calcification appeared. Additional bone salt laid down after the third injection converted many of the deposits into denser masses, which, after the fourth (Fig. 28) and

FIG. 27

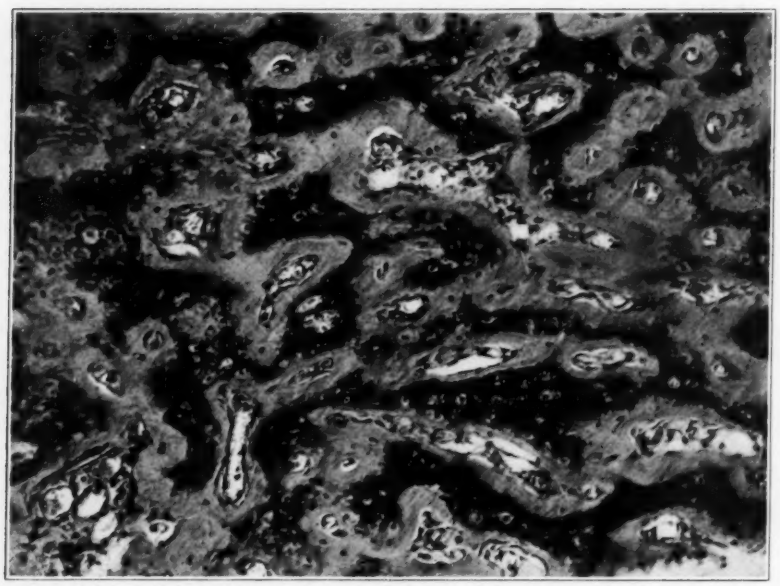


FIG. 28

Photomicrographs (× 140) show progress of calcification in the subperiosteal osteoid, following fractures in rachitic rats. Fig. 27: in untreated rachitic rat eight days after fracture the osteoid is completely devoid of bone salt; Fig. 28: shows calcification of cores of trabeculae after four daily injections of phosphate mixture, with the total phosphorus injected being thirty milligrams

fifth injections were found fusing with other closely adjacent deposits until the cores of many of the trabeculae were uniformly and densely calcified. The densest deposits of the bone salt, as seen in frozen-dried as

FIG. 29

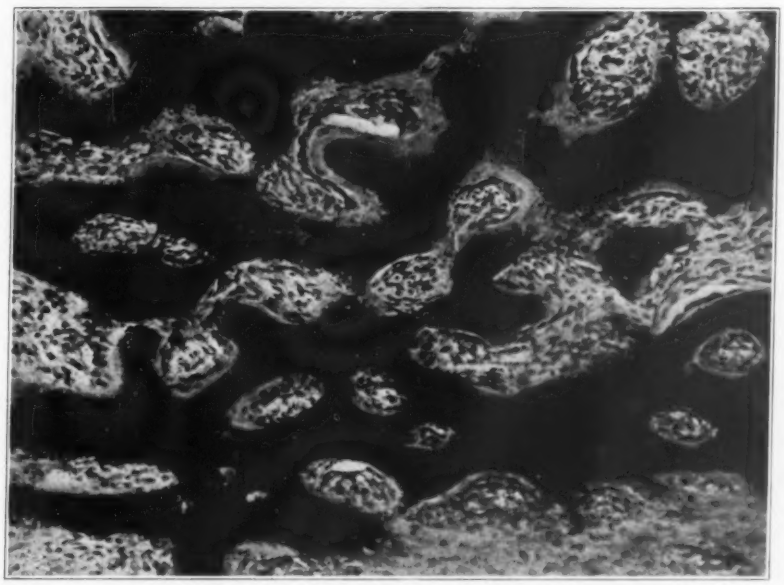


FIG. 30

per 100 grams of body weight; Fig. 29: increased density of calcification with 105 milligrams of phosphorus, as phosphate, injected in sixty hours; and Fig. 30: almost complete calcification, with thin osteoid sheaths, with 135 milligrams of phosphorus, as phosphate, injected in ninety hours.

well as in formalin-fixed tissues, were usually, but not always, in the cores of the trabeculae.

As in previously normal animals, calcification of the matrix of the callus cartilage occurs only when hypertrophic cartilage is in contact with the periosteum, or with advancing subperiosteal and subendosteal bone. In the animals in this series, in which injections of phosphate were begun on the fourth day following a fracture, the callus cartilage had become, in large part, embedded or encapsulated in dense fibrous tissue and fibrocartilage, as described in Section I. This constituted a barrier to the advance of the new intramembranous bone, so that it was not until the ninth day following the injury, or the fifth day of administration of phosphate, that contact of calcifying new bone with hypertrophic cartilage was accomplished. At this stage calcification was observed to begin in the cartilage matrix, but was limited, as in the normal animal, to the periphery of the callus cartilage where contact with new bone was established, the great mass of callus cartilage remaining uncalcified. At this time, also, there was no resumption of the removal and replacement of cartilage.

In another litter in which phosphate was given similarly until the twelfth day, it was observed that calcification in the subperiosteal intramembranous bone was greatly increased in density (Figs. 12 and 22), and that there were several areas of calcified hypertrophic cartilage in contact with the cambium layer of the periosteum. As to the penetration of the central fibrocartilaginous callus by osteogenic tissue, and as to the replacement of cartilage by bone, the animals which received phosphate from the ninth to the twelfth days of fracture healing showed little advance over those which received phosphate only to the ninth day. The newly calcified cartilage in contact with periosteum was penetrated by mesenchyme over a wide front with the formation of primary spongiosa, but there was as yet slight evidence of bone formation. Fibrous connective tissue, fibrocartilage, and chondro-osteoid separated the central callus from the areas of bone formation around the shaft, and, although these tissues were not yet being ossified, penetration by blood vessels and mesenchyme, and resorption of fibrocartilage and chondro-osteoid were under way.

Daily Injections of Phosphate from the Eighth to the Twenty-Fourth Day

As described in the foregoing sections, the callus, after four days, was so organized as to show separation of the subperiosteal and subendosteal osteoid from the central cartilaginous callus. At eight days, the fibrous connective tissue and fibrocartilage, which constituted the separating capsule, became more dense and highly developed than that seen in earlier stages. When phosphate was injected and calcification was initiated at nine days (Figs. 13, 14, 15, and 26) and continued even to fourteen or fifteen days, the bone salts were deposited almost exclusively in the subperiosteal and subendosteal osteoid, and only in such hypertrophic car-

tilage as was in direct contact with the periosteum. Limited to these areas, the deposits become very dense and widely distributed in the osteoid. Only after seven to eight days of phosphate administration (sixty milligrams of phosphorus) was there evidence of appreciable invasion and resorption, with calcification and ossification of the fibrocartilaginous callus. Continuing phosphate from the sixteenth to the twenty-fourth day of healing, when union occurred in the normal littermate, there was so much chondro-osteoid, fibrocartilage, and fibrous connective tissue to be resorbed that calcification, replacement, and ossification of the cartilage proceeded very irregularly and apparently followed the lines of least resistance to the invasion of mesenchyme. Though union finally occurred at twenty-four days, considerably in advance of untreated littermates, the structure of the new bone across the fracture line was defective. Ossifying fibrocartilage, fragments of calcified cartilage incorporated in bone trabeculae, and partially calcified osteoid were distributed in disorganized fashion between the fracture ends.

Daily Injections Begun at the Time of Fracture, or Two Days before Fracturing, Respectively

The initial stages of repair of the fracture showed no differences from those described as common to normal and rachitic animals. Between the fourth and fifth days following the injury, however, the picture differed from that just described and from that in florid rickets, in that partially calcified osteoid and calcified cartilage matrix were in communication. Encapsulation of the callus cartilage had not occurred, and there was no barrier to or lag in the invasion of the fibrocartilaginous callus by the osseous tissue arising from the periosteum. Except for deficient calcification in the newly formed osseous tissue, the process and the time relationships were essentially those seen in the normal animal.

Interruption of Calcification and Healing, and Its Prevention

In animals treated with daily injections of one-tenth molar phosphate solution there was seen not infrequently an interruption of calcification and resumption of the rachitic type of cartilage removal (Fig. 23). The following experiment was designed to study this phenomenon, and to observe the progress of calcification under conditions which assured the maximum supply of available phosphate continuously.

A litter of rachitic rats were given fractures after thirty days on the rachitogenic diet. Three days later injections of standard doses of one-tenth molar phosphate were given; half of the animals received one dose only, the remaining animals received a similar dose twenty-four hours later. The animals were sacrificed at twelve-hour intervals after the last injection.

There was found regularly at thirty-six hours, and occasionally at twenty-four hours, after the final injection a zone of calcified cartilage four to six cells wide in the rachitic metaphysis, separated from the un-

TABLE II

THE PROGRESS OF CALCIFICATION IN THE EPIPHYSEAL CARTILAGE, METAPHYSIS, CALLUS OSTEOID, AND CALLUS CARTILAGE IN RACHITIC RATS, FOLLOWING FORCED INTRAPERITONEAL ADMINISTRATION OF PHOSPHATES

GROUP	Number of Days of Healing	Total Phosphorus Injected, Mgs. per 100 Gm. of Rat Weight	Number of Hours from First Dose	Rate of Injection of M/10 Phosphate per 100 Gm. of Rat Weight	NEW CALCIFICATION			
					Epiphyseal Cartilage	Metaphysis	Callus Osteoid	Callus Cartilage
P	4.8	0	–	–	–	–	–	–
	4.1	9	3	1 c.c. per hr.	–	–	–	–
	4.2	18	6	1 c.c. per hr.	–	–	–	–
	4.4	30	10	1 c.c. per hr.	+	–	–	–
	4.7	54	18	1 c.c. per hr.	+	+	+	–
Q	12.7	0	–	–	–	–	+	–
	10.2	75	30	1 c.c. per hr. for 20 hrs.; 1 c.c. per 2 hrs. for 10 hrs.	++	+	+	–
	11.5	105	60	1 c.c. per hr. for 20 hrs.; 1 c.c. per 2 hrs. for 10 hrs.; 1 c.c. per 3 hrs. for 30 hrs.	++	++	++	+
	12.7	135	90	1 c.c. per hr. for 20 hrs.; 1 c.c. per 2 hrs. for 10 hrs.; 1 c.c. per 3 hrs. for 60 hrs.	++++	++++	++++	++++
R	8.0	0	–	–	–	–	–	–
	7.1	22.5	3	1 dose only	–	–	–	–
	7.5	22.5	12	1 dose only	–	–	–	–
	8.0	22.5	24	1 dose only	++	+	+	±

calcified epiphyseal cartilage by a narrow plate of fibrous tissue rich in blood vessels. This is the typical picture of interruption of the process of calcification and healing, and indicates that a twenty-four-hour period between doses of phosphate is barely sufficient, in the majority of instances, to keep these processes progressing continuously.

The callus of healing fractures in the same bones showed corresponding effects upon the distribution of bone salts deposited in the subperiosteal osteoid. The bone salt was found in the osteoid closest to the periosteum at twelve and at twenty-four hours after the last injection of phosphate in animals receiving either one or two injections. At thirty-six or more hours after the injection a wide layer of uncalcified osteoid was found between the periosteum and the newly calcified bone. The callus cartilage was uncalcified throughout the series.

Forced Administration of Phosphate at Maximum Possible Rate

In this experiment the progress of calcification was observed under conditions which maintained the supply of phosphate to the animal at the

greatest possible rate. Two litters of rachitic, fractured rats were injected repeatedly, each with one cubic centimeter of one-tenth molar phosphate solution per 100 grams of rat weight (three milligrams of phosphorus per 100 grams rat weight). The injections were given hourly until the occurrence of fibrillary muscular twitching, loss of posture, or convulsions, at which time, usually the twentieth hour, one cubic centimeter of one-tenth molar calcium chloride (M/10 $CaCl_2$) was given subcutaneously. The animals which survived were given additional injections of phosphate at two-hour intervals. About ten hours later the animals again went into tetany; calcium chloride was given as before, and the phosphate injections were continued at three-hour intervals. Thereafter it was possible to continue to sixty hours (from beginning the injections) without a serious number of casualties. The rats were at all times in contact with the high-calcium rachitogenic diet, which was consumed avidly. The schedule of fracture healing, and times at which the injections were given and the bones were observed is shown in Table II.

No marked differences between these animals and those given standard doses of phosphate at intervals of twenty-four hours became manifest until the sixtieth hour following the first injection. At this time there was almost continuous union between the calcified cartilage and the spongy bone in both the callus (Figs. 32 and 33) and the metaphysis. New bone in the process of deposition was calcified as rapidly as it was laid down and the osteoid everywhere in the bone and in the callus showed a heavy, uniformly distributed, though incomplete, impregnation of bone salt (Fig. 29). Transforming fibrocartilage and much of the osteoid, containing cartilage cells, was uncalcified.

At ninety hours, at which time the rats had received 135 milligrams of phosphorus per 100 grams of body weight, there was advanced calcification and healing in the epiphyseal cartilage and metaphysis (Figs. 16, 17, and 18). Dense calcification was observed in the osteoid everywhere in the original bone and in the callus (Fig. 30). Callus cartilage was as well calcified as in the normal animal at the same stage of fracture healing (Fig. 34), and the junctions between calcified cartilage and bone were continuous in both the metaphysis and in the callus. Certain areas of the chondro-osteoid contained calcium salts, but much of this tissue remained unimpregnated while undergoing resorption associated with the formation of osteoclasts. Fibrocartilage and areas of cartilage in the process of replacement by bone were calcified wherever contact was made with osteogenic tissue, but most of these tissues showed no bone salt in the intercellular substance.

IV. CALCIFICATION FOLLOWING ADMINISTRATION OF VITAMIN D

Observations were made upon the influence of vitamin D, in small and in large doses, in comparison with the effects of phosphate in inducing calcification in the epiphyseal cartilage, the metaphysis, and the fracture callus in rachitic rats.

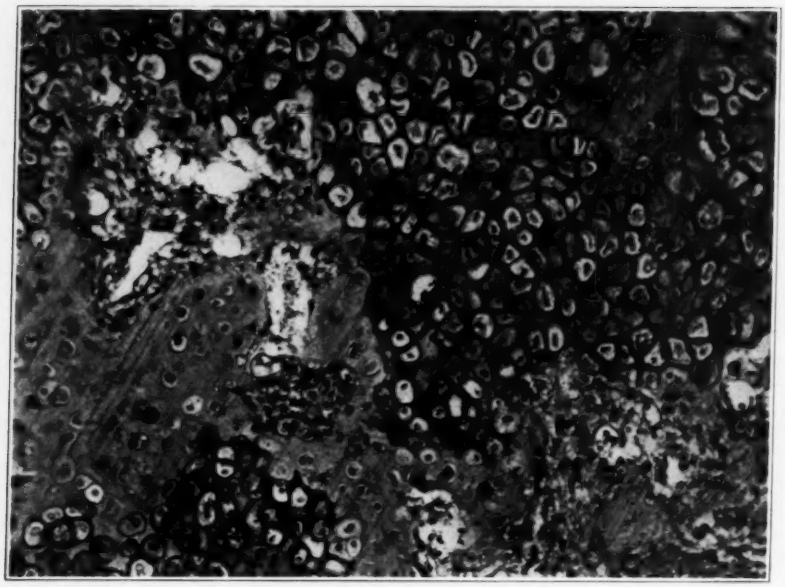


FIG. 31

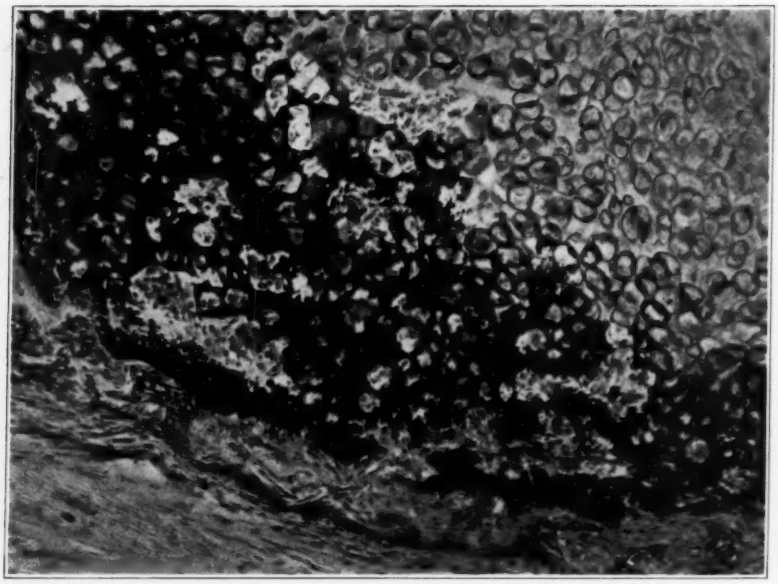


FIG. 32

Photomicrographs (× 140) show progress of calcification in the callus cartilage, following fractures in rachitic rats. Fig. 31: Shows, fifteen days after a fracture in a rachitic rat, completely uncalcified cartilage with invasion and the rachitic type of cartilage removal. Fig. 32: Subperiosteal intracartilaginous ossification twelve days following fracture in a rachitic rat which had been injected with 105 milligrams of phosphorus, as phosphate, in sixty hours. The process simulates periosteal collar and formation of ossification center in

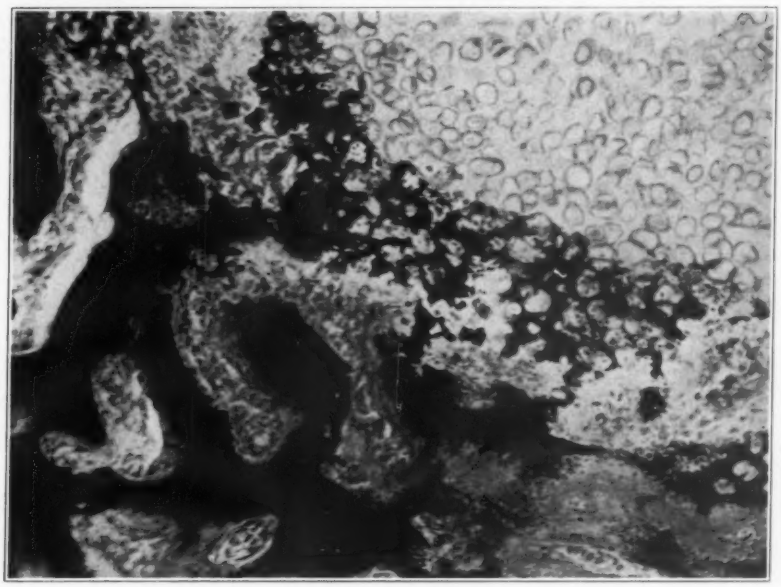


FIG. 33

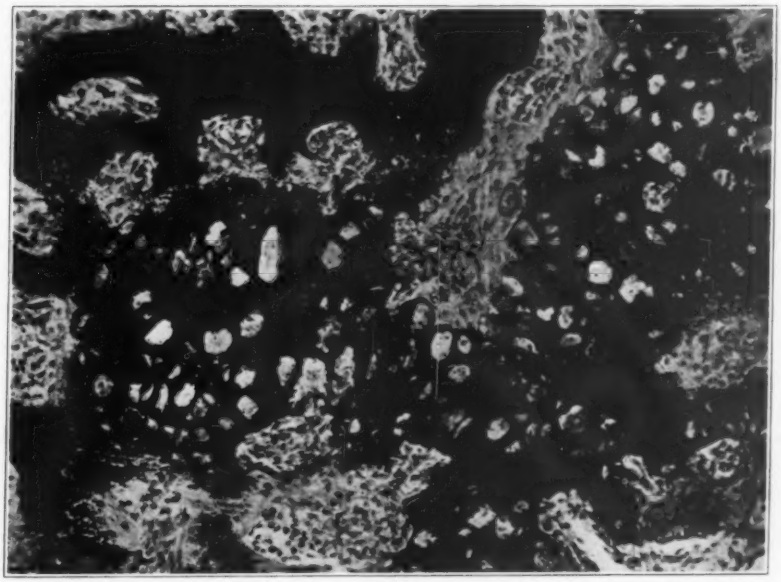


FIG. 34

embryonic bone (Bloom and Bloom). Fig. 33: Taken from the same rat as Fig. 32, at the junction of the calcified cartilage matrix and the calcified subperiosteal osteoid. It suggests primary spongiosa (compare with Fig. 10, Urist and McLean [20]). Fig. 34: Final stages of intracartilaginous ossification thirteen days after fracture in a rachitic rat which was injected with 135 milligrams of phosphorus, as phosphate, in ninety hours.

Calcification in Animals Maintained on a Vitamin-Fortified, Phosphate-Deficient Diet

It is well known that if small quantities of vitamin D are added to the Steenbock-Black diet, rickets does not appear [1,11], but bones which have a lower ash content and are smaller and thinner than normal are produced [18]. Experiments modeled after those of Shohl were undertaken with the view of learning whether the dietary conditions of reduction in phosphate intake, plus a supplement of vitamin D, would demonstrate calcification proceeding in the callus at a uniform minimum rate.

Two litters of twenty-one-day-old rats were divided into three groups to be weaned as: normal controls on the Bills diet; rachitic controls on the Steenbock-Black diet; experimental animals on the Steenbock-Black diet to which 2-per-cent. cod-liver oil had been added. The right tibiae of all were fractured at forty-nine days, and the experimental animals were divided into two groups, one of which received daily injections of 2.5 cubic centimeters of one-tenth molar phosphate per 100 grams of body weight. The normal and rachitic control groups showed normal states of calcification, on the one hand, and typical rachitic failure of calcification on the other.

The experimental groups, which were maintained on the same rachitogenic diet plus a small quantity of vitamin D, showed that the rate of calcification in growing bone and cartilage was not normal (Fig. 19). The epiphyseal cartilage was wider than normal, and the zone of provisional calcification was reduced in width. There was no chondro-osteoid in the metaphysis. The shaft was rarefied, and exhibited a considerable amount of osteoid. There was osteoid in the callus, especially at the junction between the cartilage and the advancing osseous tissue.

Calcification in the Callus in Rachitic Animals Given Large Doses of Viosterol

Two litters of rachitic rats with one-to-four-day and four-to-eight-day-old fractures were given doses of 10,000 international units of viosterol daily, by stomach tube. Two similar litters with four-to-ten-day-old fractures were given 500,000 international units daily. The bones were examined from the second to the eighth and from the fifth to the tenth day following the fractures. The animals were at all times kept on the Steenbock-Black rachitogenic diet.

Group Given 10,000 International Units of Viosterol Daily. The course and distribution of the deposition of mineral in the metaphysis and in the callus of the rachitic rats given 10,000 international units of viosterol daily were almost exactly the same as those following the administration of phosphate. The bone salt appeared in the epiphyseal cartilage and in the callus osteoid within forty-eight hours after the first dose, and increased rapidly in density and in distribution thereafter. The most striking difference seen was that after the administration of viosterol,

resorption of the chondro-osteoid of the metaphysis was much more rapid than after the administration of phosphate.

Group Given 500,000 International Units of Viosterol Daily. Viosterol in doses of 500,000 international units daily initiated calcification in the epiphyseal cartilage and in the osteoid at about the same time as did smaller doses of viosterol, or the administration of phosphate. The larger doses of viosterol, however, considerably modified the healing process in the rachitic metaphysis, and the reparative processes in the fracture callus. After three to four daily doses, the partially calcified osteoid tissue in the metaphysis, the shaft, and the callus, and the compact bone of the shaft showed widespread resorption associated with the presence of large numbers of osteoclasts. By the fourth or fifth day after administration of viosterol was begun, there was also a widespread beginning formation of new bone, associated with many osteoblasts, in the areas of resorption including the fracture callus.

V. THE RELATIONSHIP BETWEEN THE STATE OF THE BODY MINERAL STORES AND THE PROGRESS OF CALCIFICATION IN THE CALLUS OF HEALING FRACTURES

All of the above experiments were on young growing animals. The experiment here reported was undertaken to determine whether the healing of a fracture in an adult animal, with abundant stores of bone mineral, would be affected by a diet deficient in phosphorus and in vitamin D.

Twelve adult rats, which had been maintained on a stock diet for eighteen or more months, were transferred to the Steenbock-Black rachitogenic diet for a period of ten weeks. The right tibiae were then fractured and the animals were sacrificed at three-day intervals.

In all of the animals studied the spongiosa and some parts of the shaft showed very thin osteoid borders upon the bone trabeculae. Bone and the matrix of hypertrophic cartilage were densely calcified in all parts of the callus, and, except for a narrow zone of osteoid between the cartilage and the bone in the callus, there was no evidence of a mineral deficiency in the early stages of healing. The fractures united at approximately the same time as those of the controls.

DISCUSSION

As stated in the introduction to this paper, the object of these experiments was not so much to study the healing of fractures in rickets as it was to make the process of calcification and its relationship to the repair of bone accessible to analysis. For this reason the discussion will be limited to these aspects of the subject; the other essential findings reported in the paper will be included in the summary and conclusions.

In the condition of florid rickets, resulting from phosphorus deficiency, the fracture callus in rats remains completely devoid of bone salt for a period of ten to fifteen days. During this period the influence of the absence of calcification upon the healing of fractures, and the effects of

the initiation of calcification can be observed directly. At about the end of this period, calcification of the callus begins spontaneously, without simultaneous calcification in the epiphyseal cartilage and the rachitic metaphysis. Analysis of this phenomenon is a separate problem, and will be reported upon in another paper.

For the purpose of inducing calcification we have employed chiefly intraperitoneal administration of the orthophosphates of sodium, in a mixture at a hydrogen-ion concentration corresponding to pH 7.35, for the reason that the dosage supplied in this form may be accurately gauged. For purposes of comparison we have also induced calcification by the administration of viosterol, and have shown that calcification so induced does not differ from that induced by administration of phosphate.

From this and the preceding paper [20] the fact stands out that, given an adequate supply of bone minerals, calcification of the matrix of hypertrophic cartilage and of osseous tissue occurs promptly and decisively. These tissues must be regarded as already prepared for calcification, the presence or absence of bone salt depending upon the available supply of minerals.

Of especial interest is the influence of calcification, or of its absence, upon the healing process in the callus. As pointed out above, osteogenesis and calcification cannot be separated in the normal animal, so that it is impossible, from the study of normal animals alone, to determine how much the healing process itself is influenced by the deposition of bone salt in the matrix of cartilage and bone. These interrelationships have, however, been clearly demonstrated in the experiments and observations reported in this paper.

As described above, the healing process in the fractures produced in rachitic rats begins in the same way as in normal rats, and the formation of the fibrocartilaginous cartilage and the production of subperiosteal and subendosteal osseous tissue proceed just as in the normal animal, but in the absence of calcification. Evidently, then, calcification is not essential to the early stages of the healing of fractures, including the beginning of osteogenesis.

Up to the fourth or fifth day following the injury, the new tissues formed in the rachitic rat are indistinguishable from those formed in the normal rat subjected to the same procedure. But at about this time a difference appears, clearly dependent upon the absence of calcification. In the normal rat, at this time, the new intramembranous bone has gained contact with the fibrocartilaginous callus, and removal of the latter with its replacement by bone is well under way. In the rachitic rat there is a definite lag in the invasion of the callus cartilage by osseous tissue, so that active removal and replacement of the cartilage does not begin until about the ninth to the twelfth day following the fracture. In the meantime the cartilage of the callus has often become encased or encapsulated in dense connective tissues and fibrocartilage (Fig. 20).

When removal of the callus cartilage does begin, it is of the rachitic

type, duplicating almost exactly the process in the rachitic metaphysis. But the encapsulation of the callus cartilage forms a partial barrier to the process of cartilage removal, and invasion of the fibrous tissue of the callus proceeds slowly and with considerable delay.

It is difficult to determine to what extent these difficulties might be overcome in the complete absence of calcification, for the reason that in our experiments spontaneous calcification always occurred in the callus, thereby altering the picture. But it is of great interest that just as there is a lag in the healing process in the absence of calcification, so is there a lag in the response to calcification, whether spontaneous or induced.

As shown above, if calcification is induced in rachitic animals prior to the fourth or fifth day following a fracture, or before the changes characteristic of the absence of calcification have appeared, healing of the fracture proceeds in the same way and with the same rapidity as in the previously normal animal. If, however, initiation of calcification is delayed until the callus cartilage has been "encapsulated", or until the fourth to tenth days, there may be a further delay of several days until the barrier of the capsule can be overcome, and the normal type of removal and replacement of the cartilage can begin.

From the above there emerges the general principle that the initiation of healing of experimental fractures in the rat is not materially affected by dietary factors which prevent calcification, but that in the absence of calcification the healing process is both retarded and transformed into a rachitic type of response. Moreover, when conditions for calcification are restored, there is a delay in restoration of the normal healing processes, owing to encapsulation of the callus cartilage in a dense mass of connective tissue. Thus, even a temporary failure of calcification may materially retard the subsequent union of fractures. Studies of human material are now under way, in order to determine whether a similar process may be responsible for delayed union or non-union in man.

Attention has been called to the rôle of the body mineral stores as a source of calcium salts which may be mobilized under the emergency of a healing fracture. This has been shown in adult animals, whose growth is nearly static and whose bone-mineral stores are nearly maximal, by removing phosphorus from the diet. After a period long enough to present conditions of phosphorus-deficiency without complete failure in the calcification mechanism, it was possible to show that the callus of a healing fracture receives bone mineral elements mobilized from the readily available body stores, and solid union may occur at the normal time with no other source of phosphorus. In an adult rat with a well-developed skeleton, it should not be necessary to supplement the diet with mineral solely for the purpose of *enforcing the structure of the uniting fracture*, but possibly in individuals with multiple fractures and in the very young, in whom the rapidly growing bone tissue everywhere in the body demands a continuous supply of mineral, measures supporting mineral metabolism at its highest level are indicated from the earliest period after a fracture occurs.

SUMMARY AND CONCLUSIONS

1. The early stages of healing of fractures in rachitic rats resemble the same process in the previously normal animal in every respect except that of calcification. Formation of the fibrocartilaginous callus, and the production of subperiosteal and subendosteal intramembranous osseous tissue are not influenced by the complete absence of bone salt, in the first four to five days following a fracture.

2. The influence of the absence of calcification on the healing of fractures in rachitic rats begins to manifest itself by about the fourth to the fifth day following a fracture. Invasion of the fibrocartilaginous callus by the new intramembranous osseous tissue is delayed, often until the ninth to the twelfth day. This delay is comparable to the delay in initiation of the rachitic type of cartilage removal in the epiphyseal cartilage following the onset of rickets. When invasion of the callus cartilage occurs, it closely resembles the similar process in the epiphyseal cartilage in rickets.

3. Healing of fractures in rachitic rats, in the absence of calcification, may be further complicated by the embedding or encapsulation of the callus cartilage in a dense mass of fibrous tissue or fibrocartilage. This forms a barrier to the advance of the uncalcified osseous tissue.

4. Spontaneous calcification in the intramembranous osseous tissue of the callus without calcification in the epiphyseal cartilage or in the rachitic metaphysis begins in the rat at about the tenth to the fifteenth day following the injury, and leads eventually to union of the fracture by partially calcified osteoid.

5. Calcification in the osteoid of the callus, as well as in the epiphyseal cartilage and in the osteoid of the rachitic metaphysis, may be initiated by a single dose of phosphate, in a dose of 2.5 cubic centimeters of one-tenth molar phosphate mixture per 100 grams of body weight. Following such treatment the initial deposit of bone salt in the subperiosteal osteoid occurs some four to eight hours later than in the epiphyseal cartilage.

6. The distribution of bone salt in the epiphyseal cartilage, metaphysis, and callus following administration of phosphate is the same whether fixation is with neutral formalin or by the Altmann-Gersh freezing-drying method. Its localization is also identical, whether visualized by staining with silver nitrate, by *intra vitam* staining with sodium alizarin sulfonate, by observation of unstained sections with the aid of polarized light, or by the fluorescence seen in calcified areas by ultraviolet light.

7. As in previously normal animals, calcification of the matrix of the callus cartilage, following the administration of phosphate, occurs only when hypertrophic cartilage is in contact with the periosteum, or with advancing subperiosteal or subendosteal bone. When the callus has become encapsulated in the dense fibrous tissue or fibrocartilage, which forms a barrier to the advance of the new osseous tissue, there is a

delay of some days in the initiation of calcification of the callus cartilage following the administration of repeated doses of phosphate. When phosphate is given at the time of the fracture, or earlier, so that no barrier is formed, newly formed but poorly calcified osseous tissue gains contact with the callus cartilage without delay, and cartilage removal proceeds in an essentially normal fashion, with normal time relationships.

8. If phosphate is administered at longer intervals than twenty-four hours there is interruption of calcification, and resumption of the rachitic type of cartilage removal in the epiphyseal cartilage. A twenty-four-hour period between doses is barely sufficient, in the majority of instances, to maintain a continuous process of calcification. When phosphate administration is forced at the maximum possible rate, calcification in the callus is greatly accelerated, but an increased rate of phosphate administration, or an increased initial dose, has no demonstrable effect on the time of appearance of the initial calcification in the epiphyseal cartilage or in the callus.

9. A diet inadequate in phosphate, but with a supplement of two-per-cent. cod-liver oil, does not produce the gross lesions of rickets, or the manifestations of absence of calcification in the fracture callus. The distribution of the bone salt deposited in the bone and in the callus are the same whether large doses of viosterol, or phosphates are administered. Still larger doses of viosterol initiate pathological resorption of bone and of osteoid, and seriously interfere with the healing of fractures.

10. When an adult rat with a previously normal skeleton is transferred to a rachitogenic phosphate-deficient diet, calcification in the callus is adequate to produce bony union at the normal time, and there is only a very slight lag in calcification in the osseous tissue of the callus.

11. It is suggested that the observations upon the retardation of the healing process in the absence of sufficient mineral to produce calcification in the callus, and upon the delay in the initiation of a normal type of response when treatment is initiated, which results from encapsulation of the callus cartilage in a dense mass of connective tissue and fibrocartilage, may have a bearing upon certain problems of delayed union or non-union.

Our thanks are due to I. Gersh, Ph.D., of the Department of Anatomy, Johns Hopkins Medical School, for making possible the experiments upon calcification observed with the aid of frozen-dried fixation, and for assisting the work in other ways. We wish also to acknowledge the aid of A. H. Pfund, Ph.D., of the Department of Physics, Johns Hopkins University, in the preparation of demonstrations of the optical properties of bone tissue in ultraviolet and polarized light.

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CUFF RESECTION OF THE ULNA FOR MALUNITED COLLES' FRACTURE *

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From the Hospital for Joint Diseases, New York

Recent medical literature has devoted considerable attention to the so-called dislocation of the lower end of the ulna, which is not infrequently associated with improperly reduced Colles' fracture. When consideration is given to the great variation which occurs in Colles' fracture, the observer must be impressed by the similarity of the clinical picture presented by many of these patients. They invariably complain of weakness of grasp, pain or tenderness on pressure over the lower end of the ulna, limitation of rotation, and an annoying protrusion of the ulnar head (Fig. 1). The fact that this prominence is increased on pronation or supination has led to the belief that the ulnar head has dislocated. In consequence, resection of the head[1], or even reconstruction of the inferior radio-ulnar ligaments[4], has been recommended by way of treatment.

When the roentgenograms of such cases are studied attentively, one fact stands out clearly,—there has been a reversal of the normal relationships of the radial and ulnar styloids. Instead of being above, the ulnar styloid lies at a level below that of the radial styloid. Moreover, the ulnar

FIG. 1

Showing marked prominence of the right ulna, with tendency toward radial deviation of the hand.

* Received for publication October 17, 1940.

styloid projects distally so far that its shadow overlaps the shadow of the proximal row of carpal bones.

This is a matter of great importance. Normally, the additional length of the radius acts to keep the carpal bones distal to the lower end of the ulna, so that rotation is not impeded. However, when the radius is shortened by impaction, the relatively longer ulna impinges against the proximal row of the carpus and interferes with rotation. This bony block acts to cause a forward dislocation of the carpus and the radius, so that relatively the ulna appears to dislocate posteriorly.

Obviously, the indication for treatment is proper reduction, with special attention to maintaining the relative lengths of the bones. In those cases seen after firm union and deformity have occurred, a lengthening of the radius to restore the normal relationship of the bones would be indicated. However, as in other cases where equalization of bone lengths is necessary, it has been found simpler to shorten the normal bone than to lengthen the affected bone.

The following case is typical of the condition and of the therapy which has been successfully employed.

J. K., aged fifteen, first seen in the Out-Patient Department in 1935, complained of pronated feet. At that time it was casually noted that the right wrist had some radial deviation. There was no limitation of motion and no other abnormality was noted. Because of the fact that the patient made no complaint of the wrist at this time, no roentgenograms were made. The patient was not seen again until November 1939, when he returned, complaining of pain in the right wrist.

The patient stated that in 1937 he had fallen a distance of six feet and had fractured the right wrist. Treatment consisted of simple immobilization, by means of a splint, for a period of about one month. The patient apparently had had no symptoms until several months previously, when he began to have pain on motion of the right wrist.

Examination disclosed radial deviation of the hand, with a definite prominence of the lower end of the right ulna, suggesting dislocation (Fig. 1). Pronation caused a marked increase in the ulnar projection. An effort to reduce the dislocation, even by marked radial deviation, was unsuccessful, and caused much pain. Ulnar motion of the hand was limited, but all other motions were free and painless. The ulna was abnormally mobile and the hand grasp was definitely weak.

The roentgenogram (Fig. 2) disclosed a separation of the forearm bones at the distal radio-ulnar articulation. The plane of the inferior articular surface of the radius deviated sharply anteriorly and medially, so that some observers were inclined to consider it as evidence of an early Madelung's deformity. The most striking feature of the roentgenogram, however, was the discrepancy in length between the two bones. The ulnar styloid obviously projected below the level of the proximal carpal row, and the ulnar head no longer articulated with the lesser sigmoid cavity of the radius.

Since the ulna had suffered no injury, it was apparent that the disproportion between the two bones was due to the fact that the lower end of the radius had telescoped into the radial shaft at the time of fracture. In fact, it was the radius which was abnormally short, rather than the ulna which was abnormally long. Clearly the indication was to restore the relative lengths of the forearm bones, either by lengthening the radius, or by shortening the ulna. The latter was chosen as being technically less complicated.

On December 5, 1939, a two and one-half inch incision was made on the ulnar side of the forearm, just above the tip of the ulnar styloid. An oblique, subperiosteal resection of three-fourths of an inch of the ulna, one inch above the ulnar styloid, was performed. The osteotomized ends of the ulna were drawn together and wired. The wound was

FIG. 2

Roentgenogram showing interference in the growth of the lower end of the radius, apparently due to a partial synosteosis. The ulnar head projects below its normal level. The hand is in slight radial deviation.

FIG. 3

Postoperative roentgenogram. The ulna has been shortened, so that its head articulates with the radius. The so-called dislocation of the ulna has been eliminated.

closed and a plaster-of-Paris bandage was applied, with the elbow in flexion and the forearm in mid-pronation. Upon removal of the plaster on December 18, 1939, union was firm. The roentgenogram (Fig. 3) showed the ulnar head articulating normally with the lower end of the radius. The prominence of the ulna had disappeared and, clinically, there was no evidence of dislocation of the ulna. Physiotherapy was instituted to restore the normal range of motion. Examination in April disclosed normal return of painless and powerful function, with complete disappearance of the annoying deformity.

This procedure has been used with success in several such cases. The report of this case emphasizes the importance of restoring the anatomical relationship of length in adequately reduced Colles' fracture, and it presents a relatively simple method of correcting a common deformity due to improper reduction of Colles' fracture.

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EPIPHYSEAL DISLOCATIONS IN SCURVY *

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Spontaneous epiphysiolysis has been noted by many clinicians in the past. Thomas Smith, in 1876, was first to recognize the complication in a case of hemorrhagic periostitis. Thomas Barlow, in 1883, observed "crepitus, pseudoparalysis and gross deformities in two knees and two hips", which from the autopsy studies proved to be epiphyseal dislocations associated with skeletal hemorrhages. Since that time many references have been made to infantile scurvy and its associated epiphyseal luxations.

Clinically, the disorder is a state of hypovitaminosis C with a

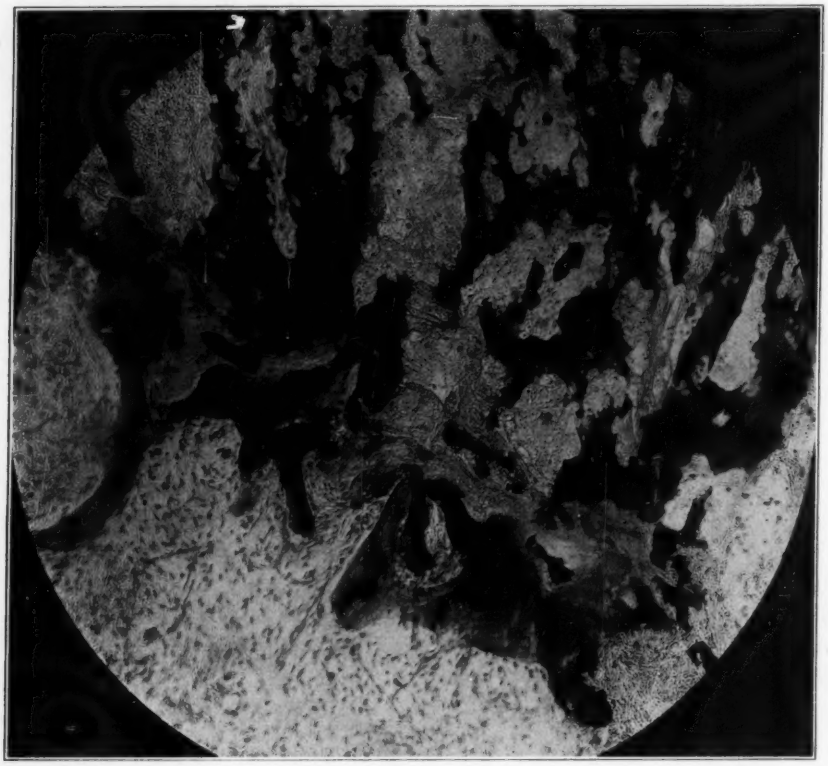

FIG. 1

High-power photomicrograph of the scorbutic lattice, showing the salient pathological features of scurvy,—highly calcified bony trabeculae with many fractures, connective-tissue invasion of the marrow spaces, and the absence of normal cellular elements. (*Reproduced by courtesy of Dr. Park and The Archives of Diseases in Childhood.*[4])

* Read before the Western Orthopaedic Association, Los Angeles Chapter, September 29, 1939.

† Service of John C. Wilson, M.D.

disposition to hemorrhages and secondary anaemia. The intrinsic factors often responsible for this state are increased body demands for vitamin C, abnormal destruction by drugs such as salycilic acid, decreased absorbability, and hypersensitivity for the vitamin or its allied foodstuffs,[3] while the responsible extrinsic causes are commonly based upon the fact that ascorbic acid is not thermostabile and that it loses potency when exposed to air[2]. These etiological factors serve to explain why scurvy is present today, our knowledge of modern preventive medicine notwithstanding.

Skeletal changes appear early in the disease, although they may pass unnoticed in mild and subclinical cases. Microscopic studies are available in only the severe cases, and have been adequately recorded[4]. Osteoclastic bone resorption is speeded up through the skeleton, but is maximal in the metaphyseal ends. The cellular elements in the marrow spaces migrate, leaving a connective-tissue frame-work (*Gerustmark*). Multiple extensive hemorrhages occur by rhexis, while cartilaginous proliferation and calcification of the proliferative cartilage remain normal.

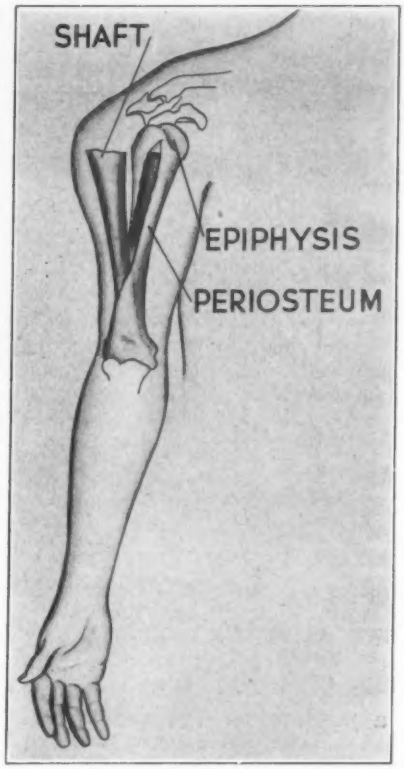

FIG. 2

Artist's drawing showing severe slipping. Note the rent and the sleevelike periosteal structure which remains for subsequent bone development. The denuded cortical shaft eventually undergoes complete resorption.

Epiphyseal fractures are a "late and very severe phenomenon" [1]. Barlow, from the pathological studies of his fatal cases made for him by Money, wrote, "I would suggest that the subperiosteal blood extravasation is the first event and the fracture the second. The extensive blood extravasation probably interferes with the nutrition of the bone, and thus the very minimum of violence, such as an ordinary movement, may lead to fracture".

While these early observations are true, it remained for more recent workers to show that the separation of the epiphysis from the metaphysis is always through the zone of calcified cartilage known as the scorbutic lattice (Fig. 1), which, in the roentgenogram, is represented by a narrow band of increased density known as the white line of scurvy or *Trümmerfeld* (Fig. 4-A). Because of its high lime content it is vulnerable to the stresses of body motion. Microscopic fractures, legion in number,

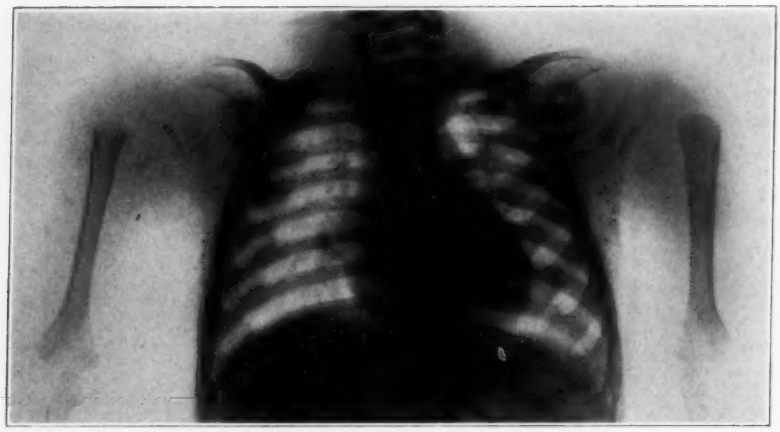

FIG. 3-A

Case 1. Roentgenogram taken March 10, 1939, showing anteroposterior view of both shoulders taken six days before admission to the Children's Hospital. Note the complete separation of the epiphyses from the shafts of the humeri, and absence of hematoma shadows.

appear spontaneously throughout the lattice (Fig. 1), very early in the clinical course of the disease, and are a consistent feature of the local pathology.

Simultaneously with the occurrence of lattice fractures, hemorrhages appear under the periosteum, and stripping takes place with little resistance; thus the last stronghold of epiphysiodiaphyseal integrity is lost. The intact periosteum maintains a "periosteal cuff" across the epiphyseal line which, clinically, is found to be a resisting factor against any displacement. If one removes the periosteum from a long bone of a young rabbit, the ease with which the epiphysis can be broken off is remarkably increased It would appear, therefore, that several factors lead to epiphyseal dislocations in scurvy: (1) spontaneous lattice fractures as pointed out by Park, and (2) subperiosteal hemorrhages, with periosteal stripping from the shaft particularly from the periphery of the epiphyseal plate (loss of the periosteal cuff).

Our case reports and serial roentgenograms illustrate the startling degree to which the infant skeleton can adjust itself to deformities by the systematic process of bone production and resorption. According to a predictable plan, new bone is laid down by periosteal bone formation on the side of the shaft toward which the epiphysis has been displaced and, to a lesser degree, on the opposite side. The epiphysis, therefore, becomes centered at the end of a newly widened metaphysis and maintains that central position throughout the secondary stage of bone resorption which ultimately shaves the bone down to its original size and shape.

The more marked dislocations are repaired by a slightly different process which is illustrated by the roentgenograms of Case 2. Figure 2 illustrates the expulsion of denuded cortical bone from its periosteal covering through a large rent. A new shaft is formed within the periosteal

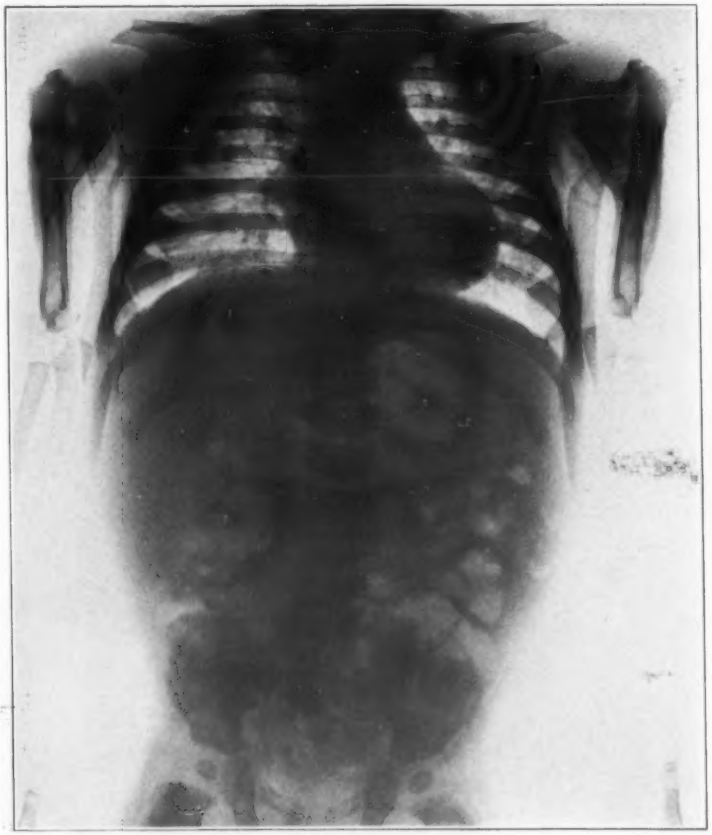

FIG. 3-B

Case 1. Roentgenogram taken April 17, 1939, four weeks after administration of ascorbic acid. The relation between the shafts and epiphyses is not altered from that of the original x-ray.

FIG. 3-C

Case 1. Roentgenogram taken June 9, 1939. Absorption of the protruding metaphysis is well advanced, and the remaining periosteal cuff is filled in quite completely. Function of the shoulder is good at this time.

sleeve, while the protruding shaft undergoes rapid resorption. For a time the new and old shafts are at an angle to each other, but in due course this angulation may be expected to disappear.

The usual locations for these epiphyseal fractures are the lower femur, upper humerus, costochondral junctions, and lower tibia, in order of frequency. Recently, Sabin found epiphyseal fractures in scorbutic monkeys, which were most marked in the lower femur and the costochondral junctions.

Some observers, particularly the roentgenologists, have endeavored to read early diagnostic signs into their x-rays, while others fail to recognize any of the bone changes as early. A history of dietary insufficiencies, the capillary fragility test, saturation determinations by the test-dose method, clinical response to treatment, coupled with oedema, spongy gums, and irritability, in a child between six and twelve months of age, will usually serve to make the diagnosis before roentgenograms become

FIG. 3-D

Case 1. Roentgenogram taken October 3, 1939. Further ossification and decrease in the angulation of the shaft.

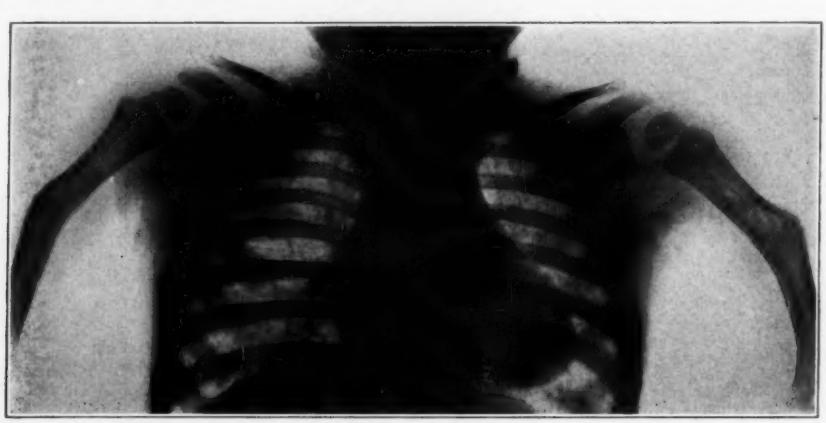

FIG. 3-E

Case 1. Roentgenogram taken January 12, 1940, shows complete clinical cure without disability.

FIG. 4-A

Case 2. Roentgenogram taken February 4, 1939. Anteroposterior views of both legs show signs of florid scurvy with typical dark (white in original X-ray) lines involving all epiphyses, especially of the lower femur and lower tibia. There are no hematoma shadows.

FIG. 4-B

Case 2. Roentgenogram taken February 13, 1939, nine days after the beginning of ascorbic-acid therapy. Lateral slipping of the femoral epiphyses can be noted. Calcification is taking place in the subperiosteal hematoma.

positive. Only one roentgenographic sign, when present, is conclusive and that is the white line with a zone of rarefaction in juxtaposition to it. The subperiosteal hematomata become calcified only after vitamin C is supplied, and a period of ten days is required for such changes to take place in the clot. A subperiosteal hematoma, when visible in the roentgenogram, is, therefore, indicative of the healing stage, and thus is a late phenomenon. Epiphyseal dislocations and subluxations likewise appear late. Calcified subperiosteal hematomata and dislocated epiphyses are not pathognomonic signs of scurvy unless they are accompanied by the scorbutic white lines. Unless this important finding be present, traumatic epiphysiolysis cannot be ruled out.

The differential diagnosis in a given case should not be difficult in the light of available diagnostic tests and roentgenographic details. Parrot's disease, congenital dislocations, and Albers-Schönberg disease, may in certain respects simulate the local findings. Traumatic dislocations can be differentiated by the absence of white lines; in other

FIG. 4-C

Case 2. Roentgenograms taken March 14, 1939. New bone is laid down by periosteal bone formation on the side of the shaft toward which the epiphysis has been displaced, and, to a lesser degree, on the opposite side. The epiphysis, therefore, becomes centered at the end of a newly widened metaphysis and maintains that central position through the secondary stage of bone resorption which ultimately shaves the bone down to its original size and shape.

roentgenographic respects the two types may be identical.

FIG. 4-D

Case 2. Roentgenogram taken October 11, 1939, shows complete clinical healing. Function is perfect.

CASE 1. Baby L., Mexican, aged eleven months, was admitted to Children's Hospital, Los Angeles, California, on March 16, 1939, with complaints of bleeding gums of four days' duration, a head cold of six weeks' standing, and marked irritability when handled. A few days prior to admission, a bilateral shoulder spica had been applied for fractures near the shoulders. The past history revealed that the child had never had orange juice, cod-liver oil, or vegetables. Examination showed an irritable, sick-looking child with a head cold. The gums were hemorrhagic, the costochondral junctions were sharp, and the shoulders showed visible deformities. The red-blood-cell count was 2,700,000, and the white-blood-cell count, 14,000. There was a moderate temperature. Treatment consisted of removing the cast and supplying a diet rich in vitamins. This included orange juice and 300 milligrams of ascorbic acid daily. General improvement was dramatic. Orthopaedic consultation was given, but no special recommendations were made for treatment, because it was agreed that the prognosis for the shoulder disabilities was good. The patient was discharged on April 17, 1939, on a prescribed diet and 25 milligrams of ascorbic acid daily. Figures 3-A, 3-B, 3-C, 3-D and 3-E show the progress as evident in the roentgenograms.

CASE 2. Baby M., female, aged nine months, was admitted to Children's Hospital, February 4, 1939, complaining of a sore left leg of two months' duration. On examination the child was described as irritable and fussy. Fresh blood was found in one auditory canal, as well as along the gum margins. The child resented any passive motions of the left lower extremity, which presented a visibly swollen ankle and knee. The red-blood-cell count was 4,900,000, the white-blood-cell count, 18,000, and hemoglobin 75 per cent. Treatment consisted of ascorbic acid in 75-milligram doses daily. A high vitamin, high caloric diet was prescribed. The child was discharged on February 18, 1939, in good general condition. The healing process is shown by progressive roentgenograms (Figs. 4-A, 4-B, 4-C and 4-D).

CONCLUSIONS AND SUMMARY

1. Scorbutic epiphysiolysis is rare in infancy.

2. Microscopic lattice fractures and the loss of the "periosteal cuff" are the factors responsible for the slipping.

3. A natural realignment of shaft and epiphysis takes place following the use of vitamin C, which leads to recovery without deformity or disability.

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POLYOSTOTIC FIBROUS DYSPLASIA WITH CUTANEOUS PIGMENTATION AND CONGENITAL ARTERIOVENOUS ANEURYSMS

A Case Report

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Fibrous dystrophy involving multiple bones has been differentiated in recent years from localized and generalized osteitis fibrosa cystica.[1, 9] Lichtenstein believes it represents a distinct clinical entity and has proposed the term "polyostotic fibrous dysplasia". Three further cases have appeared under this designation.[7, 10, 13] A syndrome with similar bone pathology, cutaneous pigmentation, and precocious puberty in females has been described by Albright and his coworkers[2, 3] and others.[4, 8, 12, 14, 15, 16] A case has been studied which presented predominantly unilateral fibrous replacement of bone, and cutaneous pigmentation. Multiple arteriovenous aneurysms were located in those extremities showing the most marked bone involvement.

CASE REPORT

History

G. B., male, aged nineteen years, was admitted to Temple University Hospital on the Service of Dr. Temple Fay on April 13, 1940, because of protrusion of the left eye of two weeks' duration. There had been brownish discoloration of the left upper eyelid, lacrimation, and pain shooting to the vertex on closing the eye. On admission, these had diminished, and the proptosis was receding.

The past medical history revealed the usual childhood diseases. The family history was negative. The patient came of northern European stock. Early development was normal. A triangular brown area was present on the back from birth.

At two and one-half years of age trauma to the left forearm was followed by enlargement and bowing. At five years, after a minor injury, protrusion of the left forehead was first noted. At this period, following roentgenographic study, a diagnosis of "bone cysts" was made. The left femur was fractured twice during the sixth year, resulting in deformity of the hip and a persisting limp. At eleven years there was a fracture of the neck of the left femur. Bone deformity of the left upper extremity progressed gradually, chiefly from the eighth to the twelfth years, with an increase in the diameter of the arm. Minor trauma usually preceded the development of the enlargements, which frequently were painful at the outset.

Prominence of the veins of the left upper extremity had been present since early childhood, and the parents had become aware of unusual vibrations palpable over it when he was about ten years of age. At that time a roentgenologist stated that the boy's heart was greatly enlarged.

A biopsy was made from the proximal end of the left radius at another hospital when he was eleven years of age. The pathological diagnosis was osteitis fibrosa cystica. On that basis, exploration of the neck was carried out, and a small parathyroid gland re-

FIG. 1

Photograph showing deformities of left side of body and proptosis of left eye.

moved, which was reported histologically normal. Subsequently, a series of roentgen-ray treatments of the parathyroids was given. The patient felt that there had been little progression of his deformities since the parathyroidectomy.

Puberty had occurred at about the normal time and sexual desire was normal. The cardiorespiratory, gastro-intestinal, genito-urinary, and nervous systems were essentially negative. Growth and weight gain were normal. Despite limitation of activity due to the deformities, he had been graduated from high school. His intelligence was well above average, and he had developed sufficient manual dexterity to undertake drafting as an occupation.

Physical Examination

The patient was somewhat underdeveloped, measuring five feet five inches in height and weighing 113 pounds. Obvious deformity was confined almost entirely to the left side (Fig. 1). Bony enlargement of the left frontal region and left lower jaw was present. The left arm was greatly increased in diameter and showed a posterior bow; the forearm was increased in diameter in its upper third, and the hand was greatly distorted. The deformities and resulting ankylosis rendered this extremity practically useless. In the right upper extremity, slight swelling of the proximal third of the arm, of the elbow, and several small bones of the hand was the only apparent involvement. The left hip was prominent, showing coxa vara deformity and partial ankylosis. The diameter of the upper third of the thigh was increased. On the left there were genu valgum and 10 degrees' back-knee, with cavus deformity, and equinus of the foot. There was three centimeters of shortening on the left, the patient walking with an obvious limp on this side. The right lower extremity was normal. A marked lumbar scoliosis, convex to the right, was present.

The skin was pallid. It was smoother and warmer over the left arm and thigh. In the same areas the hair was more sparse and downy. Distribution of hair was normal otherwise, except for scanty growth on the face. A triangular, coffee-colored, non-elevated area of pigmentation was located on the lower posterior thorax, just left of the midline (Fig. 2).

The left eye was proptosed with lacrimation and conjunctival oedema. The extraocular movements were limited in all directions. Fundal examination showed beginning oedema of the left disc. Perimetry revealed only a relative central scotoma on the left. Vision: left eye $^{6}/_{15}$; right eye $^{6}/_{12}$. Examination of the nose and ears was negative. Audiometer readings showed bilateral mixed deafness. Bárány findings were normal. The teeth were normally developed. The thyroid gland was not enlarged.

The heart was greatly enlarged, with a forceful apical thrust. The apex beat was

palpable in the fifth interspace, eleven centimeters from the midsternal line. The left border was percussed at the anterior axillary line in the fifth interspace. A harsh basal systolic murmur was best heard in the second left interspace. On compression of the left axillary and femoral arteries, the murmur diminished perceptibly, together with slowing of the pulse rate from eighty to sixty per minute. This "bradycardiac phenomenon" was abolished by one-thirtieth of a grain of atropine sulphate given intravenously. It was accompanied by a reduction in the amplitude of cardiac pulsations, as revealed by fluoroscopy. The latter showed enlargement of all chambers of the heart with a computed volume of 925 cubic centimeters. The electrocardiogram was normal. The blood pressure in the recumbent position was 150/90 in the right arm, and 160/0 in the left. Thrills and continuous bruits with systolic accentuation were present above the left clavicle and throughout the left arm, especially over the larger arteries. There was also a thrill and bruit over the left femoral artery just below the inguinal ligament. The veins of the left arm were greatly dilated and tortuous, the larger ones showing systolic pulsations (Fig. 3). Oscillometric readings showed abnormally great amplitude of pulsations over the left arm, forearm, and thigh. Venous-pressure determinations gave equivocal results. Sodium-cyanide circulation times were: right arm, 15.8 seconds; left arm, 11.2 seconds; and right femoral vein, 9.2 seconds. Skin-temperature readings over the left arm averaged from 1 to 3 degrees Fahrenheit higher than on the right. Sweating occurred more readily over the left arm than elsewhere following pilocarpine (The development of blue color in a starch-iodine mixture was used as an indicator).

FIG. 2

Photograph of back showing pigmented area to the left of the midline.

The abdomen and genitalia were essentially normal. Rectal examination was negative. Neurological examination revealed nothing significant except an equivocal Chvostek's sign.

Roentgenographic Findings

Examination of the skeleton revealed most of the bone structures to be involved by an unusual process. The bone changes were all of the same type but differed in degree and extent, and, apparently, in duration at various sites. While the lesions were predominantly on the left side of the body, there were similar, but less well-advanced, changes on the right side, exclusive of the lower extremity.

The skull (Fig. 4) showed marked thickening of the frontal bone with marked irregularity in density. The frontal sinus had been replaced by expanded, coarsely trabeculated bone. The bone structures along the floor of the anterior and middle

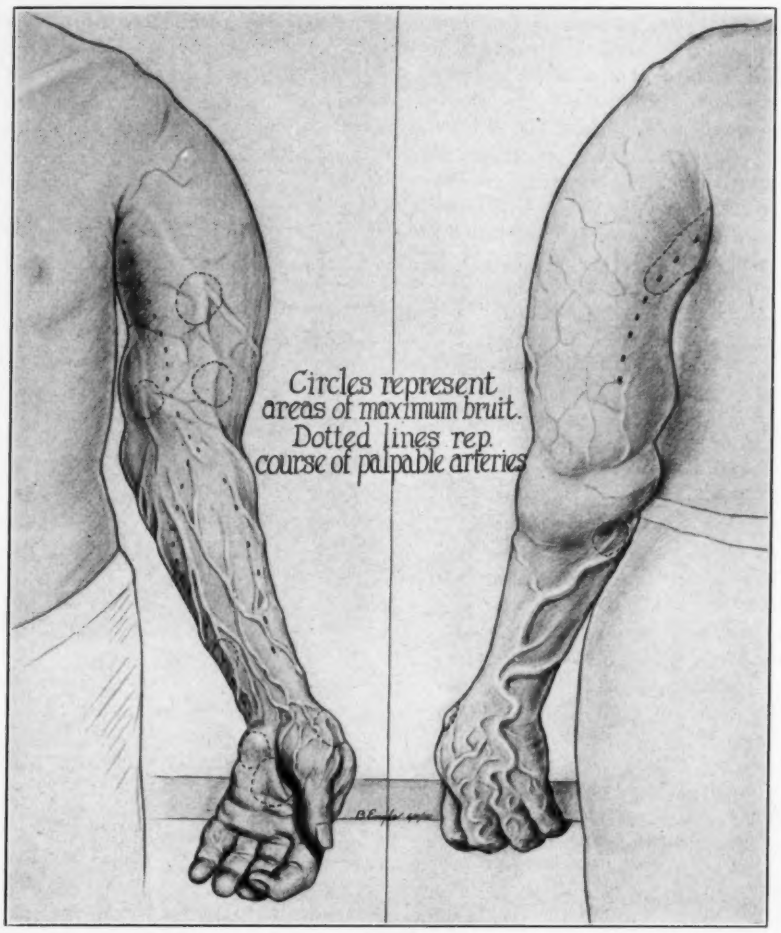

FIG. 3

Drawing of left upper extremity showing dilated veins and indicating course of palpable arteries with the sites of maximum bruit.

fossae were involved, but the posterior part of the skull appeared normal. The involvement on the left side was much more extensive.

The left humerus (Fig. 5) was greatly expanded, and the cortex was thin and irregular. Bowing was present. The medullary portion of the bone showed coarse, irregular trabeculations of increased density, intermixed with less dense areas simulating cysts, but stereoscopic study failed to reveal any evidence of a cyst wall.

The hands (Fig. 6) showed involvement, more marked on the left side. The bones were expanded and the cortex greatly thinned, but there was no evidence of perforation of the cortex at any site. Marked irregularity in density of these bones was noted. The trabeculations were prominent and ran at all angles, thus simulating cysts, but no true cysts could be demonstrated.

The pelvis (Fig. 7) revealed marked asymmetry with the distortion confined to the left side. Softening and molding of the pelvis in the region of the acetabulum had deformed these structures, and the acetabular fossa was very shallow. There was marked shortening of the left femur contributed to by the marked angulation between the neck and shaft of the femur, and by the coxa vara deformity.

In addition to the areas illustrated, the left lower extremity, the right upper extremity, the mandible, several ribs, and several vertebrae also showed involvement.

Laboratory Data

In determinations on finger blood from both hands the hemoglobin was 9.5 grams per 100 cubic centimeters and the red blood cells 4,230,000 per cubic millimeter. Differential and platelet counts, sedimentation rate, prothrombin, and coagulation times were normal. There was no Bence-Jones protein or other abnormal finding in the urine. Blood and spinal-fluid serology, and the tuberculin test were negative. Basal metabolic rate, gastric analysis, Mosenthal test, urea clearance, and glucose tolerance all were within normal limits.

Oxygen saturation of venous blood * in the right forearm was 34.8 per cent., in the left forearm, 90.2 per cent.

No anterior pituitarylike hormone or estrone was detected in two forty-eight-hour urine specimens. In two twenty-four-hour specimens there were 7.0 milligrams and 4.0 milligrams respectively of androgens, calculated as androsterone, by the method of Callow, Callow, and Emmens.[5]

(In several determinations between September 8, 1932, and August 20, 1934, at another hospital, the serum calcium had been reported as between 9.2 and 12.5 milligrams per 100 cubic centimeters; and the phosphorus between 2.3 and 3.9 milligrams per 100 cubic centimeters.)

In weekly determinations the serum-calcium level was between 7.0 and 9.0 milligrams per 100 cubic centimeters; the phosphorus between 3.6 and 4.6 milligrams per 100 cubic centimeters. The phosphatase activity ranged between 37.6 and 64.0 Bodansky units, except for an isolated value of 132 units. Blood sugar, urea nitrogen, non-protein

* Oxygen-saturation determinations were done by Dr. Robert H. Hamilton, Department of Physiological Chemistry.

FIG. 4

Roentgenogram showing characteristic bone lesions. (See also Figs. 5, 6, and 7.)

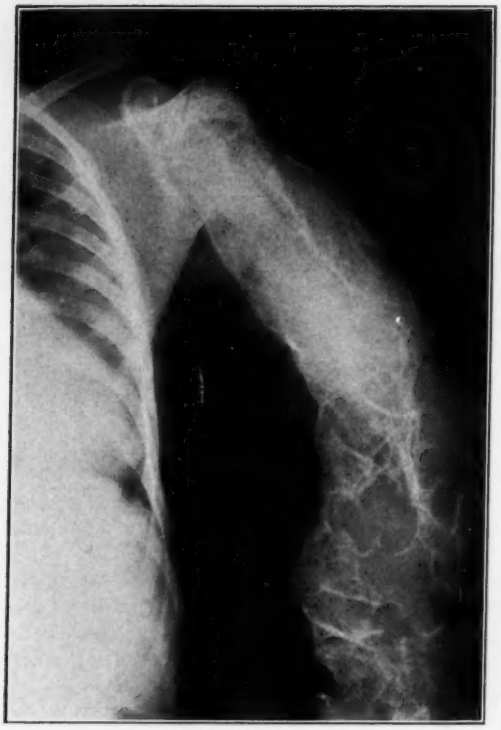

FIG. 5

nitrogen, chlorides, cholesterol, and cholesterol esters were within normal limits.

Two six-day calcium-balance studies were essentially normal. On a calcium intake of approximately 0.1 grams daily the output averaged 0.01 grams in the urine, and 0.14 grams in the faeces daily. On a daily intake of approximately 0.9 grams the output averaged 0.01 grams in the urine, and 0.30 grams in the faeces daily.

The smear from a sternal puncture revealed no gross abnormality of marrow cytology.

Course in Hospital

The patient was ambulatory throughout his stay of nine weeks. The proptosis receded spontaneously, and the eye had regained normal appearance by the end of the third week. The extra-ocular movements were also restored to normal, and the oedema of the left disc was no longer present. During the first week the blood pressure in the right arm remained about 160/100; from that time until discharge it was consistently about 120/70. The spinal-fluid pressure was twenty millimeters of mercury in the first week and eight millimeters in the third week. No adequate explanation was found for these changes. There was no other evident alteration in his clinical condition.

Treatment

The hypochromic anaemia failed to respond to ferrous sulphate in adequate dosage. Ertron (200,000 U. S. P. Vitamin D units daily) was given from May 27, 1940, to June 15, 1940, and dicalcium phosphate (3.0 grams daily) from June 4, 1940, to June 15, 1940, with no significant effect on the serum level or urinary output of calcium.

Pathological Report

Biopsy specimens were taken from the greater trochanter of the left femur, from the right ulna, and right humerus June 8, 1940,* and from the area of pigmented skin. Grossly, the bone specimens consisted of elastic, gritty tissue covered by a very thin layer of cortex. Sections presented very similar microscopic pictures (Fig. 8). Where the compact cortical bone was intact it appeared thin, and in some places was degenerating. Despite the loss of normal cortical bone there was very little evidence of osteoblastic activity of the periosteum. It was impossible to distinguish between the medullary and spongy portions. Both these zones were replaced by a fibrous matrix in which were embedded irregular spicules and fragments of degenerating bone, imperfectly calcified bone, and osteoid tissue.

Bone spicules in all stages of disintegration were found. The location of this ma-

* Bone biopsies were performed by Dr. John Royal Moore, Department of Orthopaedic Surgery.

FIG. 6

FIG. 7

terial showed little relation to the presence of giant cells. The spicules identified as bone were often heavier, more irregular than normal, with spotty calcium deposition and a fairly normal surrounding membrane of osteoblasts in specially stained tissue. The greater proportion of differentiated tissue, however, was composed of irregular spiculelike pieces of osteoid or fibrous bone tissue. This material showed little inclination to calcify.

FIG. 8

Photomicrograph of a section of bone biopsy from left greater trochanter showing typical fibrous dysplasia with poorly formed and degenerating bone.

The entire background consisted of a fibrouslike tissue of varying density composed in a few areas of cells loosely and haphazardly placed, showing the morphology of fairly young osteoblasts. More generally, the cells were definitely spindled, the nuclei relatively pyknotic, and arranged in bundles resembling more adult fibrous connective tissue. The presence of large amounts of collagen was demonstrated by special staining. There were fewer blood channels in this tissue than in normal spongy bone, though the tissue was by no means avascular. In the majority of the fields osteoclasts or giant cells were lacking. In some areas, however, these cells were present in sufficient numbers to suggest the histology of a benign giant-cell tumor, and usually contained more than a dozen nuclei which were arranged in the center of the cell without pattern. Mitotic figures were found in none of the cells. No lipoid deposits or significant collections of inflammatory cells were seen. The whole suggests a diffuse fibrous dysplasia which has upset normal bone architecture and growth. There was both osteogenesis and osteolysis, yet neither process appeared to be normal. Comparison of sections of this tissue with those of a bone biopsy taken eight years ago in another hospital presented essentially the same picture, except that in the latter more of the fibrous cells appeared to be younger, suggesting a more active phase of the disease.

A section made of the biopsy removed from the pigmented skin showed an entirely normal histology except for an abnormal amount of pigment which was concentrated in the basal cells and deeper layers of the stratum spinosum. This resembled the histology encountered in sections of the pigmented areas from cases of von Recklinghausen's neurofibromatosis.

The section of the parathyroid gland taken at about the time of the first bone biopsy showed a moderate secondary hyperplasia. The chief cells were normal in size, though increased in number, and showed a tendency to an acinic arrangement. Fat and oxyphil cells were lacking. The lack of oxyphil cells may be due to the age of the patient when the biopsy was taken. The rest of the picture is entirely compatible with a diagnosis of secondary hyperplasia as described by Castleman and Mallory.[6]

DISCUSSION

The findings in this case eliminated generalized osteitis fibrosa cystica, osteitis deformans, Ollier's dyschondroplasia, and neoplasm. To explain the bone disorder there remained the condition variously called osteodystrophia fibrosa, regional fibrocystic disease, osteitis fibrosa in multiple foci, etc. Lichtenstein's descriptive term, polyostotic fibrous dysplasia, seemed peculiarly appropriate for this typical case in view of the widespread distribution of the lesions. The condition is characterized as follows:

1. *Clinical Picture:* Deformity, pain, and limp usually appear in childhood or early adult life. Pathological fractures, frequently with delayed union and malunion, bowing of weight-bearing bones, and coxa vara are common. Involvement tends to be unilateral. The process progresses slowly over many years, eventually becoming static.

2. *Roentgenographic Findings:* Any of the bones may be involved, the most frequent being the femur, tibia, humerus, and radius, in the order named. The diaphysis and metaphysis are regularly affected, the epiphysis more rarely. The shaft is expanded, with the cortex markedly thinned. There are numerous irregular, apparently trabeculated areas of decreased density, commonly misinterpreted as cysts. Sometimes, especially in the radius or tibia, diffuse evenly distributed porosity with a paper-thin cortex may be seen.

3. *Chemistry:* The serum calcium is normal or slightly elevated. Calcium-balance studies have, for the most part, yielded normal results. The serum phosphorus is normal. Significant elevation of the phosphatase level is frequent.

4. *Pathology:* Thinning of the cortex is obvious grossly, the medullary cavity being occupied by solid, resilient, gritty fibrous tissue. Cysts are infrequently found. Microscopically, relatively avascular fibrous tissue replaces the cancellous bone and marrow. Trabeculae of imperfectly calcified new bone and osteoid tissue are scattered through this fibrous matrix. Periosteal deposition of new bone is not obvious, and the endosteal surface is eroded. Areas of giant cells may be found and occasionally islands of hyaline cartilage. There is no histological evidence of inflammation or neoplasia. The histological picture of the bone probably is not specific for this disease. In biopsy sections of cases of osteitis fibrosa cystica, benign giant-cell tumor, and in some instances, perhaps, osteitis deformans, the histology may be similar.

Instances of similar lesions involving a single bone have been described.[9] These may represent a monostotic variant or a stage in the evolution of the disease. There is also the possibility that lesions in other bones have been overlooked.

This case differed in one particular from the typical case as described above. The serum calcium was persistently low during hospitalization. Eight years before, prior to parathyroidectomy, and during two years thereafter it was reported normal. This might suggest that mild hypoparathyroidism had developed. It is more likely that considerable variation in the serum-calcium level may occur in the individual case, depending on the activity of the bone lesions.

It seems worthy of mention that many of the cases in the literature were also subjected to exploratory operation for parathyroid adenomata and some to parathyroidectomy before the nature of the condition was recognized.

The skin pigmentation relates this case to those of Albright and his coworkers, McCune and Bruch, and others [2, 3, 4, 8, 12, 14, 16] which had bone lesions apparently representing polyostotic fibrous dysplasia, together with cutaneous hyperpigmentation, and precocious puberty in females. As in this case, there was a tendency to unilaterality, the skin and bone manifestations both being more marked on the same side. No endocrine dysfunction was observed in the few male cases reported by the above authors. However, polyostotic fibrous dysplasia was recently described in a male sixteen years of age with feminine secondary sexual characteristics.[13] A large hemorrhagic cyst of the skull was an atypical finding in this case. Albright, Scoville, and Sulkowitch[3] suggested that the unilaterality in these cases might best be explained on the basis of a disseminated neurological lesion, to which the endocrine dysfunction is secondary. No evidence was found in the present case for either endocrine or neurological disturbance.

The diagnosis of multiple congenital arteriovenous aneurysms in the left upper extremity, with the strong probability of similar communications involving the left femoral artery and vein, was substantiated by the following findings:

1. Thrills and bruits over the left upper extremity and left femoral artery.
2. Oxygen saturation of 90 per cent. in venous blood from the left forearm.
3. Higher skin-temperature readings over the left upper extremity.
4. Greater amplitude of oscillometric readings over the left arm, forearm, and thigh.
5. Bradycardiac phenomenon.
6. Cardiac enlargement.

This appears to be the first reported instance of polyostotic fibrous dysplasia with such a complication. Although hemihypertrophy of extremities may occur with arteriovenous fistulae, the latter probably played a minor rôle in producing the extreme degree of involvement of the left upper extremity and femur. The association of this congenital vascular anomaly with a disorder of bone, likewise a mesodermal derivative, strongly suggests that the two may have some congenital factor in common. This supports the view of Lichtenstein that polyostotic fibrous dysplasia results from a congenital disorder of the bone-forming mesenchyma. The varied findings which have been associated with it in the reported cases may, for the present, we believe, logically be assumed to represent further manifestations of the defect in the germ plasm responsible for the bone pathology.

SUMMARY

1. A case of polyostotic fibrous dysplasia with cutaneous pigmentation in a male is presented, together with the diagnostic criteria of the disorder.

2. The presence of arteriovenous communications on the side of the predominant bone involvement is advanced as additional evidence for a congenital etiology of the condition.

3. Recognition of the nature of this osseous dystrophy may prevent unnecessary surgery and possible sacrifice of normal parathyroid glands.

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A STUDY OF PARALYTIC SCOLIOSIS BASED ON FIVE HUNDRED CASES OF POLIOMYELITIS *†

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The purpose of this report is to review the findings in a series of 150 patients with paralytic scoliosis, and to analyze some of the mechanical factors involved in the development and treatment of these cases. An attempt has been made to determine the reasons for the development of the curvature, and its possible prevention and treatment. The follow-up period has extended back twenty years with a rather even distribution of cases each year except 1937, in which there was a poliomyelitis epidemic within the state.

GENERAL CONSIDERATIONS

Scoliosis has developed in 150 patients, or 30 per cent., of the 500 cases of poliomyelitis studied to date. This seems an unusually high percentage, and the authors hope to show some of the reasons for it, keeping in mind that a revision of this percentage may have to be made as the study continues. A Scoliosis Clinic at the Crippled Children's Hospital has been established, and all cases of lateral curvature of the spine have been registered. Sixty-two per cent. of them have been caused by

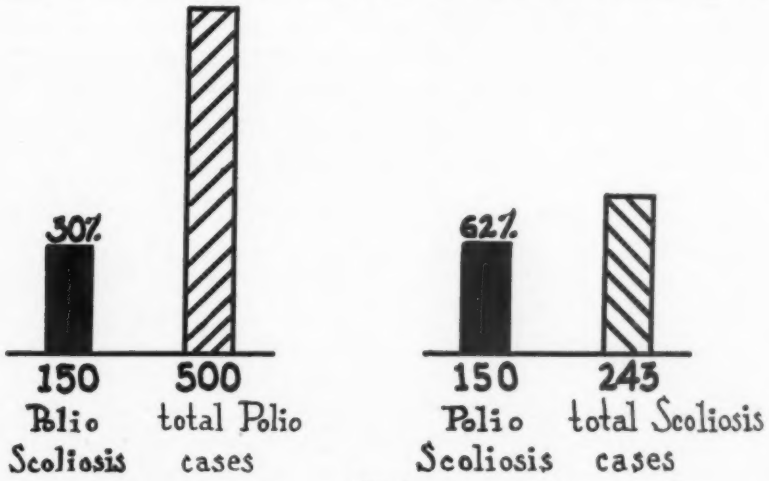

FIG. 1

* Read at the Annual Meeting of the American Orthopaedic Association, Kansas City, Missouri, May 7, 1940.

† This work has been made possible by a grant from The National Foundation for Infantile Paralysis, Inc.

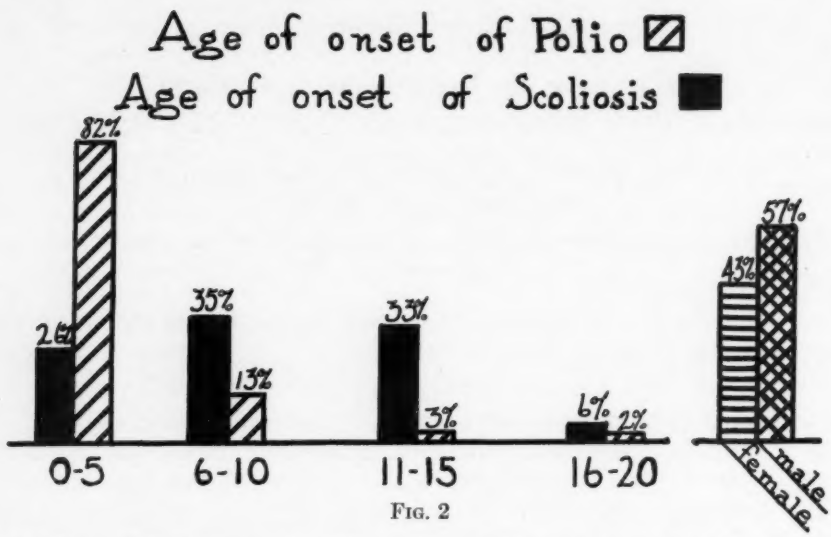

FIG. 2

infantile paralysis (Fig. 1). Figure 2 shows the age of onset in all the poliomyelitis cases, and the approximate age of the patient when the scoliosis developed. There was a latent period, before the development of the curvature, which ranged from a few months to twelve years, but in this delayed-development group the patients presented little if any trunk involvement. Here it is felt that other mechanical factors such as weak hip muscles or unequal leg length played the more important part.

It has been said that scoliosis will not progress after growth ceases; this the authors have found to be true of the idiopathic type, but not true of all the paralytic cases in this series. Four cases were found in which the curvature developed after the age of sixteen, though repeated measurements showed no further growth. In one a severe scoliosis developed at about twenty years of age. In a review of 500 poliomyelitis cases, forty-five, or 13.5 per cent., had some upper-extremity paralysis, and in thirty-six, or 81 per cent., of these scoliosis developed. Four hundred and thirty-five patients, or 87 per cent., had some lower-extremity involvement, and in ninety-six, or 22 per cent., of these scoliosis developed. One hundred and fifty-seven, or 31 per cent., of these patients had trunk involvement, of whom 135, or 86 per cent., showed a scoliosis. Seven per cent. of the patients with structural scoliosis had extremity involvement without any demonstrable trunk paralysis. There were twenty-one cases with paralytic involvement of the trunk, but without scoliosis two years or more after the attack of poliomyelitis. This group is particularly interesting and will be dealt with in more detail later. In the total cases examined, only seventeen patients have had a complete recovery from their muscle paralysis, and of these the extremities were involved in thirteen and the trunk in four.

Very few of the patients with paralytic scoliosis had adequate early treatment, as 69 per cent. had no recumbency or a period of less than

six weeks after the onset of the disease. Twelve per cent. were recumbent from six to twelve weeks; 14 per cent., from three to six months; and only 5 per cent., six months or more. Back supports were employed in only one out of every five, and 59 per cent. had no support at any time. Sixteen per cent. had support before the spinal deformity was detected, and twenty-five per cent. received support only after the scoliosis was obvious. It is of interest that severe deformity developed in many of the latter cases. It is the authors' belief that efficient support of a progressive scoliosis is very difficult, and with the high thoracic curve it is virtually impossible. With a lumbar curve, the spine may be supported in a plaster or celluloid jacket, or occasionally with a crutch type of brace.

Thirty patients had been fitted with canvas corsets, usually before becoming ambulatory, and standing roentgenograms, taken with and without the corset, showed these supports were usually ineffective. Various types of back braces were employed, but it was not infrequently found that the curvature extended above the edge of the brace so that the lack of support was obvious. In most of the cases with progressive curves, the inadequacy of any type of supportive treatment could be demonstrated by the standing and supine roentgenograms. The accordionlike deformity which developed in many of these patients was remarkable, and head traction often demonstrated an amazing degree of correction. Figures 3-A and 3-B show a child who was easily lengthened eight inches by head traction, being manually lifted.

While it is the authors' general impression that scoliosis develops more rapidly during active growth periods, other factors—such as a short

FIG. 3-A

FIG. 3-B

Photographs of patient, aged eight, in sitting position. Note the accordionlike characteristic as she is lifted manually by the chin and occiput.

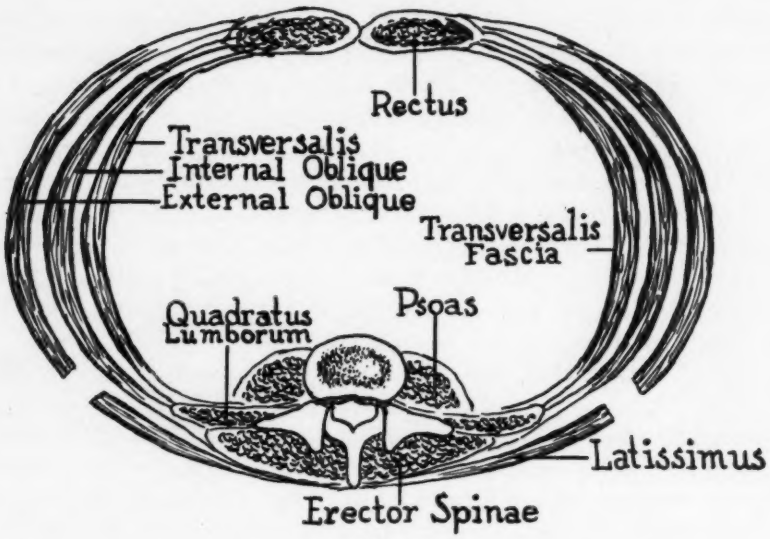

FIG. 4

NOTE: The names of the muscles shown on this drawing do not in all cases coincide with the names generally used in *The Journal:*
Rectus = rectus abdominis;
Transversalis = transversus abdominis;
Internal oblique = obliquus internus abdominis;
External oblique = obliquus externus abdominis;
Psoas = psoas major;
Erector spinae = sacrospinalis.

period of recumbency following the disease, the build of the patient, and the severity of the original paralysis—will also influence the degree of the curve. Severe curves developed in all of the cases with marked asymmetrical paralysis, though in 14 per cent. progression was arrested and corrected by turnbuckle jacket and fusion treatment.

The postpoliomyelitis scoliosis developing in a young child is usually a progressive affair. Therefore, efforts should be directed toward prevention of the scoliosis as far as possible. The authors believe that any patient with a trunk paralysis should have a minimum period of six months in absolute recumbency. Physiotherapy may be instituted after the muscle tenderness has subsided and, if intelligently used, will undoubtedly aid, but if not intelligently used it may strengthen the strong muscle groups and not affect those which are paralyzed.

PHYSIOLOGICAL CONSIDERATIONS

As pointed out by Jones and Lovett many years ago, lateral flexion and rotation of the spine do not occur as separate movements; so, when the spine is bent laterally, a certain amount of rotation automatically occurs, and the bodies of the vertebrae are rotated toward the convexity of the curve.

The majority of the lateral curvatures begin at the junction of the thoracic and lumbar regions. The thoracic region of the spine normally has very little motion in any direction, not only because the thoracic cage

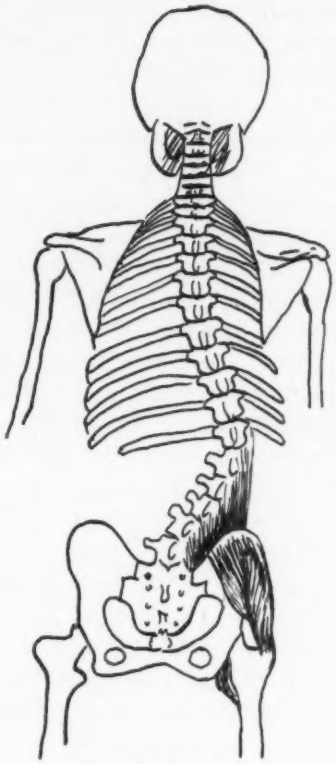

FIG. 5

Paralysis of the iliopsoas occurred in 103 cases, and of the gluteus medius in 120 cases.

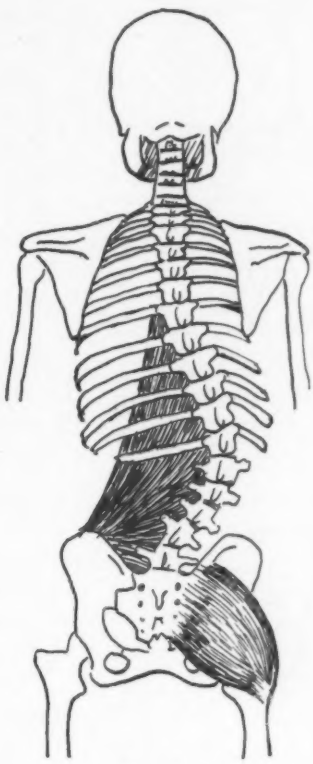

FIG. 6

Paralysis of the obliqui abdominis occurred in 126 cases, and of the gluteus maximus in eighty-five cases.

is attached in this region, but also because of the conformation of the individual vertebrae in this portion of the spine and the absence of any muscles permitting flexion in the thoracic cage. Posteriorly the sacrospinalis group of muscles produce extension throughout the entire spine. In the lumbar region the quadratus lumborum, psoas major and minor, and the abdominal muscles play a very important part in producing paralytic scoliosis (Fig. 4). Mackenzie has pointed out that the abdominal muscles do not produce flexion or rotation of the trunk, but with this the authors do not agree. These muscles are, however, primarily for support of the abdominal wall and for use in the physiological processes of respiration. Contraction of the rectus abdominis, Mackenzie points out, does not cause physiological relaxation of the sacrospinalis group of muscles. These abdominal muscles give increased strength to visceral support and are useful as the antagonists of the diaphragm muscles. The authors are convinced that the principal muscles flexing the spine are the rectus abdominis, internal and external obliques, as well as the iliopsoas and quadratus lumborum. If the muscles producing the patient's spinal move-

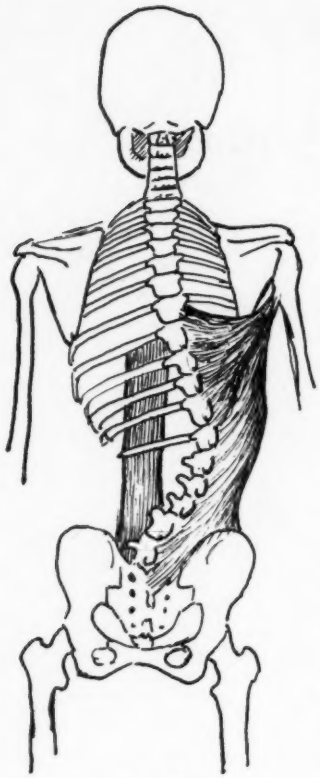

FIG. 7

Paralysis of the rectus abdominis occurred in seventy-four cases, and of the latissimus dorsi in sixty-three cases.

ments act normally, there is a normal range of motion. If the flexing muscles can act only unilaterally they will, with the exception of the iliopsoas, produce a lateral deviation and rotation of the spine with the concavity toward the strong side. The iliopsoas will produce a convexity toward the strong side. In this study lumbar paralytic scoliosis with abdominal-muscle involvement alone has been found, although occasionally the same curve has developed without abdominal-muscle paralysis. It would be particularly interesting to know whether the type or degree of curve that will routinely develop from a spinal or abdominal paralysis can be predicted. It may be of some value to compare the spinal potentialities for movement with those found in an extremity. In the extremities various movements are produced by one set of flexors, rotators, and extensors, but in the spine these movements are produced by two sacrospinalis groups, two iliopsoas muscles, two quadratus lumborum muscles, etc.,—one set on each side of the body. This construction of two groups of flexors and two groups of extensors and rotators greatly increases the complexity of the whole scoliosis problem. The deformity, therefore, is always dependent upon the muscle pulls plus the individual bone conformation of the particular vertebrae involved, and *these factors are present even though the paralytic patient is recumbent.* The authors have seen curves produced through this muscle imbalance even though the patient was recumbent and properly splinted. Added to this is the great factor of gravity whenever the patient resumes the upright position. Therefore, deformity is produced by the pull of the stronger opposing group on one side or other of the body plus gravity. Steindler states that where the great rotators of the spine are lacking, the curvature may develop during recumbency, and he believes the external oblique is one of the most important rotators. The abdominal oblique muscles are the stabilizers of the rib cage, which enable it to swing laterally whenever the arm and shoulder girdle movements and the leg and pelvic movements produce an asymmetrical pull, and this pull gives rise to a rotary spinal deviation. The pull in one direction of the serratus posterior inferior, which is the chief opponent of the abdominal oblique and opposite transversalis colli, may be the stabilizing influence against this side twisting.

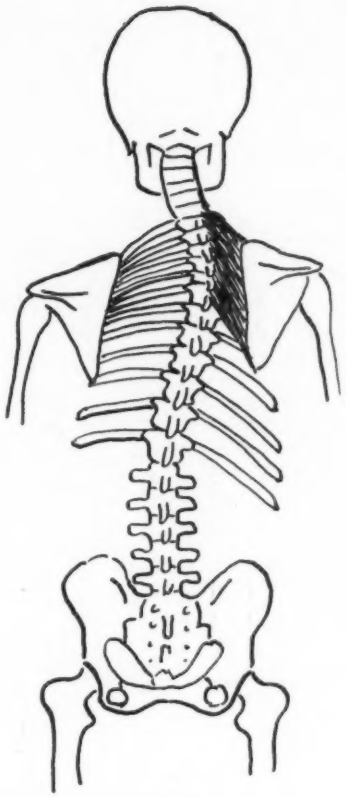

FIG. 8
The rhomboids were paralyzed in forty-two cases.

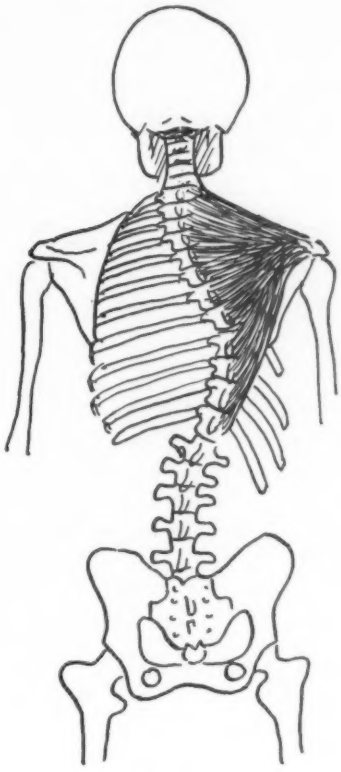

FIG. 9
The trapezius was paralyzed in twenty-eight cases.

MUSCLE GROUP INVOLVEMENT

Mayer made the observation some years ago that a fixed pelvic obliquity may be caused by contracture of the spinal muscles, of the abductor or adductor muscles about the hip, or by a combination of these, and Irwin has recently written on this same subject. Certain cases of pelvic obliquity, arising from contracture of the abductor group of muscles of the hip, with a distinct lowering of the pelvic crest and subsequent curvature of the spine have been noted in this study. This spinal curvature becomes obvious when the limbs are placed parallel,—the attitude assumed in weight-bearing. Only two cases have been found presenting fixed pelvic obliquity from this cause, which is somewhat at variance with the findings of Mayer and his associates. The authors are inclined to think this adductor contraction is frequently secondary, rather than primary, in cases of paralytic curvatures of the spine. They have attempted to classify the development of the paralytic progressive curve in relation to the muscle pull, and feel that the stronger muscles always tend to produce deformity, a fact not so well recognized in spinal deformity as in the paralytic deformity of the extremities. In addition to the pull of the asymmetrical

FIG. 10

The curve was toward the sound side in twenty-seven cases of paralysis of the sacrospinalis, and in twenty-three of the forty-five cases of paralysis of the deltoids.

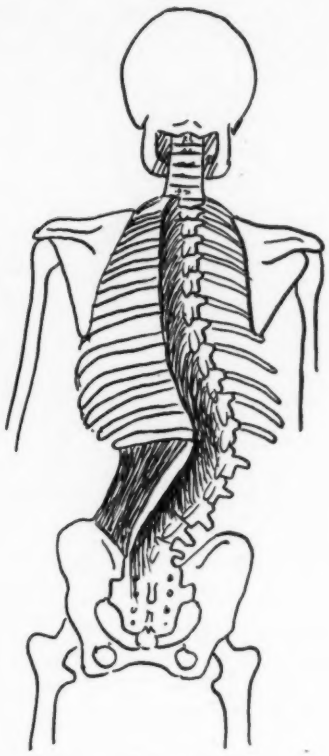

FIG. 11

The muscle pull was asymmetrical in nineteen of the cases of paralysis of the sacrospinalis, and in twenty-eight of the forty cases of paralysis of the quadratus lumborum.

muscle forces, the uncomplicated inequality of leg length is sometimes sufficient to initiate the process. Unequal leg lengths was felt to be the primary factor in twenty-two cases in this series.

The cases have been divided into two groups: (A) having the convexity of the curve toward the stronger muscle groups, and (B) having the concavity of the curve toward the stronger muscle groups.

In Group A unequal pull of the iliopsoas was present in twenty-nine of 103 cases, and as far as could be determined was the principal factor in producing the curve (Fig. 5). The same type of curve was found in fourteen patients of 120 with gluteus medius paralysis, and in eleven patients out of eighty-five with gluteus maximus paralysis (Fig. 6). Convexity toward the sound side may result from paralysis of the latissimus dorsi and this spinal deformity was found in thirty-four of a total of sixty-three paralyzed muscles (Fig. 7). This apparently is a very important type of paralysis, and in six cases the latissimus dorsi appeared to be wholly responsible for the development of the curvature. A similar curve was

found in rhomboid (Fig. 8) and trapezius (Fig. 9) paralysis,—that is, a pull toward the sound side in thirty-six of a total of forty-two affected rhomboids and in the twenty-eight paralyzed trapezii. Deltoid paralysis (Fig. 10) gave rise to a curve toward the sound side in twenty-three of forty-five cases of paralyzed deltoids.

Group B. Lowman has repeatedly emphasized the importance of asymmetrical abdominal-muscle paralysis. Thirty-one cases in this series presented the concavity of the curve toward the side of the strong abdominal muscles. In this abdominal group there were a total of 200 paralyzed muscles with 126 involving the obliques and seventy-four the recti (Fig. 7). A curvature with the concavity toward the sound side was noted in the cases of paralysis of the sacrospinolis, which was a very important factor in the development of many of the dorsal curves. The muscle pull was asymmetrical in nineteen cases of the forty-six with the sacrospinalis muscles involved and in twenty-eight of forty cases of paralyzed quadratus lumborum muscles; the concavity was always toward the sound side (Fig. 11). The other twelve patients presented symmetrical paralysis of the quadratus lumborum.

TYPES OF CURVES IN PARALYTIC SCOLIOSIS

In this study the curves produced have been divided into total and combined curves. The total curve of the long thoracolumbar type was found in seventeen cases. In the combined group, with primary and secondary curves, there were 133 cases. In this study of paralytic scoliosis 110 patients returned for roentgenograms, taken with the patient both supine and standing, which have been carefully reviewed and correlated with the muscle paralysis.

The primary curve in this combined type can be divided into three classes: the thoracic, thoracolumbar, and lumbar. There were sixty-three patients in the thoracic group. These curves have been divided roughly into those extending from the first to the ninth, from the third to the eighth, and from the fifth to the twelfth thoracic vertebrae, and an attempt has been made to analyze each type for the muscle pull or pulls producing it. In the thoracic-curve group the scoliosis is apparently produced by the upper extremity and back paralysis. Of these sixty-three cases, thirty-three had the convexity to the right and thirty to the left, showing that the muscle paralysis is about equally divided on the two sides of the body. In the second class of combined curves, the mid or thoracolumbar curve, extending from approximately the sixth to the third lumbar vertebrae, there were twenty-nine cases. These apparently were produced primarily by a combination of abdominal and upper or lower extremity involvement, especially the latissimus dorsi. In the lumbar or lower type of curve, extending roughly from the tenth thoracic to the fourth lumbar vertebrae there were forty-one cases. These were primarily produced by paralysis of the latissimus dorsi, abdominal muscles, iliopsoas, or the abductor muscles about the hips. Beevor's sign

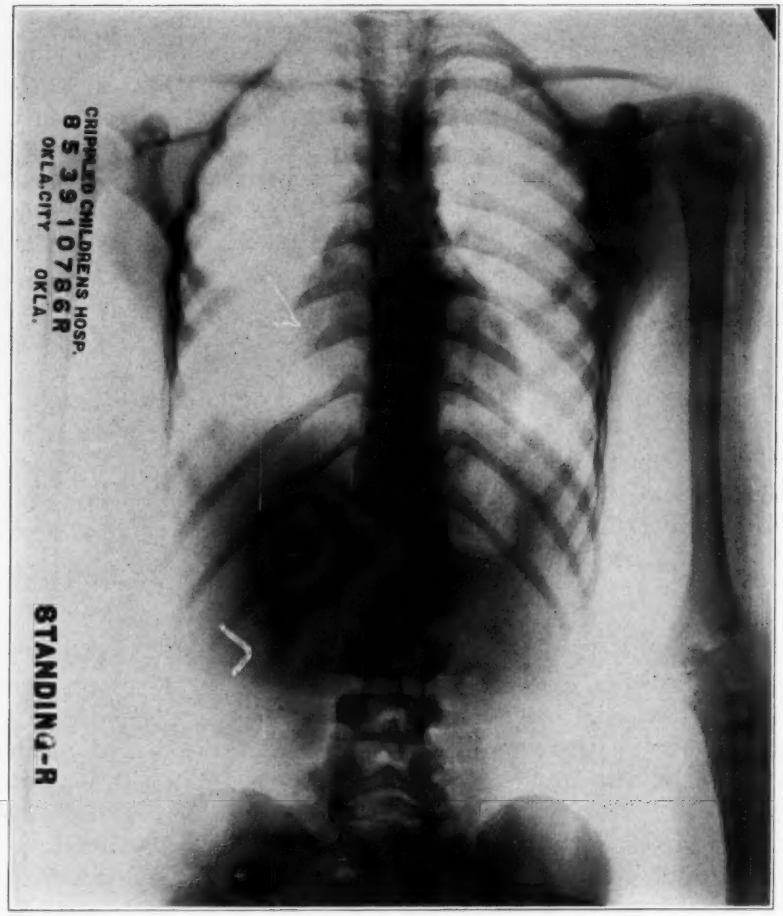

FIG. 12-A

Roentgenogram of patient with complete abdominal paralysis and a marked. lordosis, but no scoliosis. Fig. 12-A: Anteroposterior view of patient standing.

is a valuable one,—that is, there is normally no movement of the umbilicus on raising the head from the supine position, but with paralyzed or weakened abdominals the umbilicus shifts its position as tension is placed upon these muscles. In this series of cases, Beevor's sign was unfortunately not routinely observed, but every one of the nineteen patients in whom it was found presented abdominal weakness or paralysis.

Occasionally a double primary curve may result from a rather extensive paralysis. One such patient had adequate muscle paralysis to explain the two primary curves, and in addition had the two usual compensatory curves,—a total of four instead of the usual three curves.

TRUNK PARALYSIS WITHOUT SCOLIOSIS

In the total series of cases, twenty-one patients have not developed scoliosis and yet present definite trunk paralysis. These are not included

FIG. 12-B

Lateral view of same patient in supine position.

FIG. 12-C

Lateral view of patient standing.

in the 150 cases of paralytic scoliosis, but constitute a very interesting and informative group. Without exception the trunk paralysis in each of these cases was almost completely symmetrical. In this group patients were found with complete abdominal paralysis and a marked lordosis without lateral curvature (Figs. 12-A, 12-B, and 12-C). Conversely, in over 500 unselected cases of poliomyelitis, there was not a single case with marked asymmetrical trunk paralysis in which a scoliosis did not develop. In this small group of twenty-one cases, there were fourteen patients who had been followed for two years or more after the attack of poliomyelitis, but had not developed a curve; so the future development of scoliosis in these cases may be regarded as possible though improbable (Fig. 13). If the trunk can be balanced by the remaining symmetrical groups, it brings up the question of utilizing this principle in treating cases of muscle imbalance by methods not ordinarily used. Correction of paralytic scoliosis has not been treated as effectively as paralytic deformities elsewhere. In the foot, for example, muscles are transplanted or sectioned in combina-

FOURTEEN CASES

Trunk Paralysis without Scoliosis

Left Right

14	13	12	11	10	9	8	7	6	5	4	3	2	1	case number	1	2	3	4	5	6	7	8	9	10	11	12	13	14
13	30	5	4	4	7	3	4	4	3	5	20	6	7	age	7	6	20	5	3	4	4	3	7	4	4	5	30	13
11	6	2	2	2	2½	1½	2	1	3	1½	4	4		onset age	4	4	1½	3	1	2	1½	2½	2	2	2	2	6	11
P	FG	GF	N	N	N	GN	N	N	N	N	P	N	F	Erector Spinae	F	N	O	N	N	N	GN	GN	G	N	N	GF	FG	P
P	O	GF	N	N	N	GN	N	N	P	N	P	F	GN	Quadratus Lumborum	GN	N	O	N	P	N	GN	GN	GN	N	GN	GF	O	P
F	N	GF	N	N	N	GN	N	N	N	N	G	N	G	Latissimus Dorsi	G	G	N	N	N	N	N	GN	N	N	G	GF	N	F
G	FG	GF	GN	N	N	GN	N	N	N	N	N	N	G	Rhomboids	G	G	N	N	N	N	N	GN	N	N	GN	GF	F	G
T	FG	GF	G	F	N	G	G	N	G	FG	O	N	N	Rectus Abdominus (upper)	N	G	O	FG	G	N	G	G	N	N	F	GF	FG	O
P	FG	GF	G	G	FG	G	N	N	G	PF	O	N	N	Rectus Abdominus (lower)	N	N	O	PF	G	N	N	G	N	G	F	GF	FG	P
O	N	GF	FG	N	N	F	G	N	T	G	O	G	N	Oblique Abdominal (upper)	N	G	O	G	P	N	G	G	N	F	PF	GF	N	P
P	N	GF	FG	N	N	F	P	FG	P	G	O	F	N	Oblique Abdominal (lower)	N	G	O	G	P	FG	P	P	FG	F	FG	GF	N	P
T	G	N	F	N	O	O	F	FG	O	N	O	F	N	Iliopsoas	N	G	T	FG	O	O	FG	N	G	N	F	N	G	P
P	O	G	FG	N	P	O	PF	P	O	N	T	P	N	Gluteus Medius	N	F	P	FG	O	F	G	N	PF	O	FG	G	O	P
G	N	O	N	N	N	N	N	N	N	N	G	N	O	Deltoid	F	N	T	N	N	O	N	N	N	N	N	G	N	T
F	N	O	N	N	N	N	N	N	N	N	N	N	N	Trapezius	N	N	G	N	N	N	N	N	N	N	N	G	N	F

N=Normal G=Good F=Fair P=Poor T=Trace O=Absent

FIG. 13

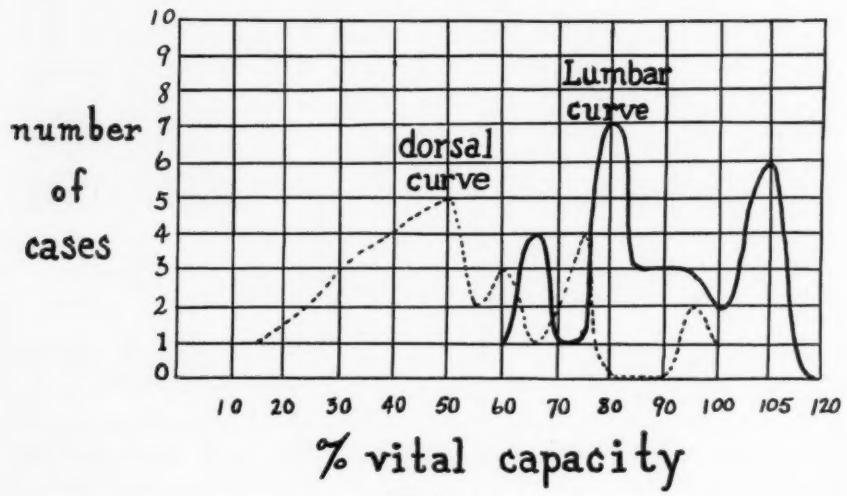

FIG. 14

tion with joint fusion to prevent recurrence of the deformity. May deformities which are produced by the vicious pull of asymmetrical muscle groups be corrected through myotomies, transplantations, etc., rather than by simply fusing the spine?

OBESITY

About 30 per cent. of the total number of patients became more or less obese following their poliomyelitis. The increase in weight was frequently quite sudden and usually began about four months after the onset of the disease. A number of our patients have had a low basal

metabolic rate, and an endocrine disturbance is probably present. This added weight is naturally a disadvantage to any patient with weak trunk muscles, and is an added burden on the spine.

VITAL CAPACITY

In sixty-two of the patients with paralytic scoliosis, vital-capacity readings were obtained, using the DuBois method with a Collins spirometer, and it was found that those with scoliosis of the thoracic region produced the lowest vital-capacity reading, while those with curves in the lumbar region consistently had a higher reading. The vital capacity of the sixty-two patients varied from 15 per cent. to 111 per cent. with an average in the thoracic region of 56 per cent. and in the lumbar region of 88 per cent. (Fig. 14). Those patients in whom the curve involved the rib cage presented compression and displacement of the lungs and heart, often to an alarming degree. The patients with an extremely low vital capacity are naturally an easy prey to upper respiratory and lung infections and many of them are obviously poor surgical risks. The use of empyema blow bottles and toy balloons into which the patients blew ten or twelve times daily, frequently caused marked improvement in the vital-capacity reading. In a number of cases these simple methods more than doubled the vital-capacity reading in from one to three months.

METHODS OF TREATMENT

Every effort should be made to improve the general condition of these patients by a careful regimen of muscle training, sun baths, and chest-expansion exercises. Attention should also be paid to diet, either for increasing or reducing the weight of the patient. Several of these patients also had a preoperative course of glandular therapy to improve the lowered basal metabolism and lessen the tendency to obesity.

Lowman states that the loss of the integrity of the abdominal muscles is closely related to the development of paralytic scoliosis, and has suggested the use of fascial strips. The authors' observations have confirmed his findings that fascial strips give a feeling of security to the patient, and have a stabilizing effect upon the remaining trunk muscles and pelvis. The patients have expressed themselves as feeling much more comfortable, and by better balance they have been relieved of the feeling of "toppling over backwards". A fascial sling has also been used in a case with paralyzed rhomboids; the strips were extended from the spinous processes to the vertebral border of the scapula, but the authors' experience has been so limited that they cannot draw definite conclusions regarding its benefit. The stripping of muscles and fascia from the spinous processes and laminae has been reported, but the authors have had no personal experience with these procedures. As far as can be learned through personal communications, these results have been rather transitory.

When an optimum general condition has been reached and an evaluation of the muscle paralysis made, it can be assumed that the development

of a scoliosis will simply be a matter of time, if the paralysis is asymmetrical, but the opposite will be true if there is a fairly symmetrical involvement. This latter type of case should be studied very carefully for a long period of time for the possible development of asymmetry and a late scoliosis. Supine and standing roentgenograms are taken of all patients at three-month intervals. If there is a definite progression of the curve, fusion has been recommended as the only method that will effectively stop the progression. The younger the patient, the greater is the necessity

FIG. 15

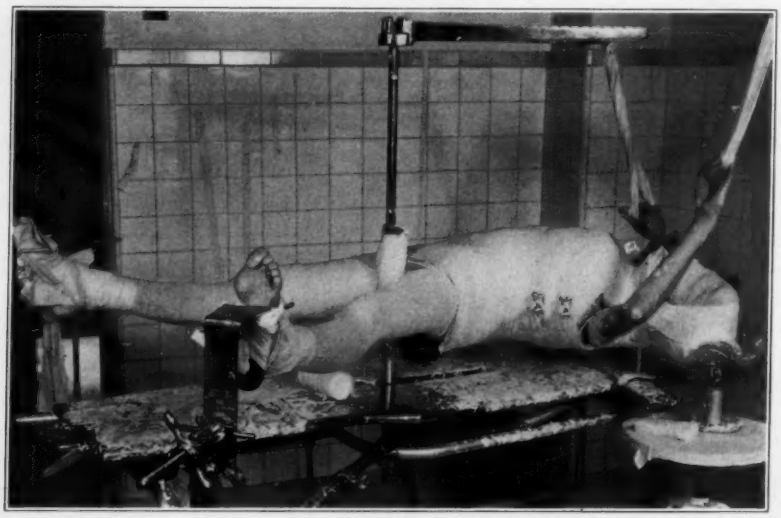

FIG. 16

Application of plaster jacket with patient's trunk bent toward the concave side of the primary curve always straightens to some extent the secondary curve.

for corrective jacket and fusion, provided the paralysis is grossly asymmetrical. If the patient is at the end of the growth period and roentgenograms show little or no mobility of the primary curve, fusion may not be necessary for the prevention of further deformity. However, other factors will influence the patient in the choice of a fusion operation,—such as appearance, balance, and stability, any of which may make fusion desirable.

Only 14 per cent. of the patients have had fusion operations, but a great many more are being prepared for fusion because it is felt it offers the patient a great deal of benefit. The operative treatment of scoliosis depends upon the age, and the mobility and extent of the curve, as well as the general condition of the patient. The extent of the primary curve is calculated by the method of Risser and Ferguson, and the Risser type of jacket is usually employed for correction.

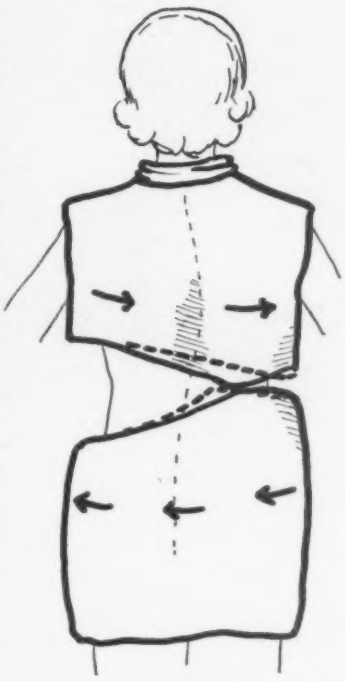

FIG. 17

Method employed for derotating the spine.

The jacket is applied with the patient's trunk bent toward the concave side of the primary curve, because in this way the secondary curves can be partially or completely straightened (Figs. 15 and 16). This also has the advantage of considerably derotating the spine because the primary curve apparently acts as a unit. After maximum correction has been obtained by the usual wedging method, another modification has been used. The hinges and turnbuckles are removed and by exercising great care not to lose lateral correction, the upper part of the jacket is rotated on the lower part so as to bring the shoulder on the convex side of the curve forward. (Fig. 17). Marked cosmetic improvement has resulted from this manœuvre, and the fusion operation has been thereby greatly simplified. The operation is performed through a window in the original plaster. Figure 18-A shows a roentgenogram of a child of eight, presenting a thoracolumbar type of curve straightened (Fig. 18-B) in the usual fashion, though moderate rotation can be noted. The spine was then derotated by bringing the right shoulder forward, and, as can be seen, complete derotation was obtained (Fig. 18-C).

Fusion was performed by a modified MacKenzie Forbes technique. Additional bone is usually used and may be taken from the tibia or ilium. In the mid-thoracic region a little more extensive dissection has been done, using the transverse processes and rib shavings as additional bone.

It has not been possible to derotate completely all the spine, so that

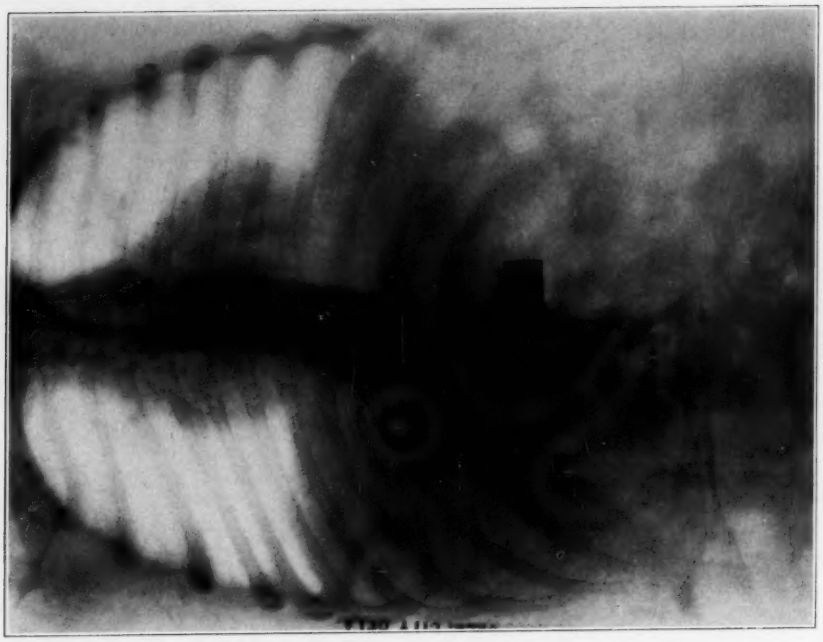

FIG. 18-B
Correction in turnbuckle type of jacket.

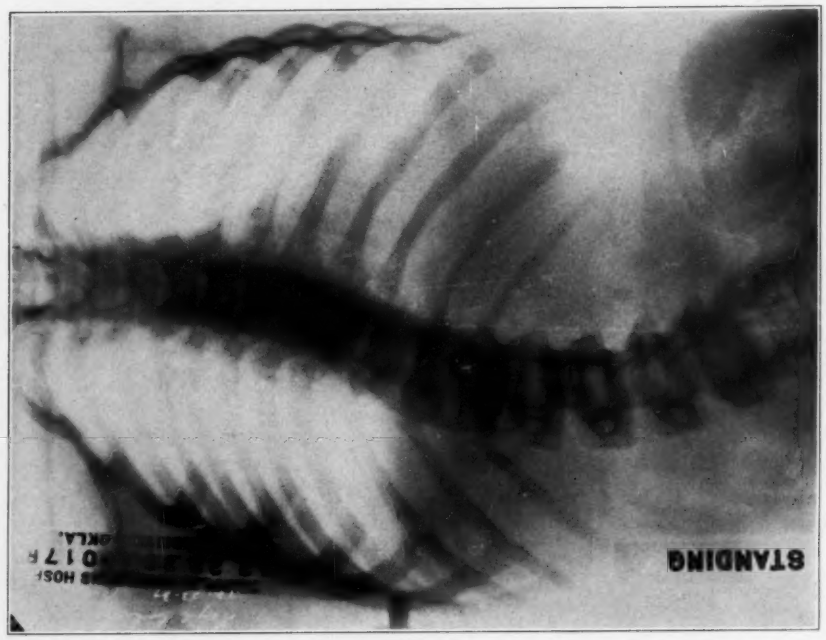

FIG. 18-A
Paralytic thoracolumbar type of curve.

some of the older, stiffer, more deformed patients may retain a fairly marked razor back after all possible correction. With this type of deformity some of the deformed ribs have occasionally been removed at the time of fusion and used as bone grafts. This method was described in 1927 by Armitage Whitman.

Pain resulted from impingement of the scapula on the deformed ribs in some of the older cases in whom fusion was not necessary. These cases were frequently benefited by rib resection. The vital capacity was not affected because apparently the lung on that side had been previously so compressed as to be of little functional value.

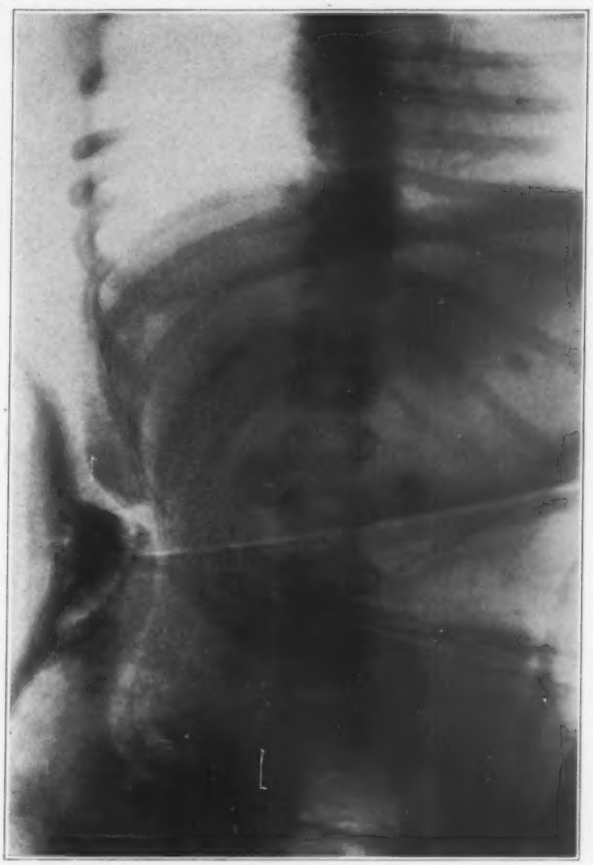

FIG. 18-C

Derotation obtained as noted in the text.

Because of the deforming effect of some of the active muscles in patients with asymmetrical scoliosis, the question has been raised as to whether or not some type of muscle operation would not offer an opportunity of lessening this vicious force. Using the foot as an example, it is known that, despite adequate and solid fusion, deformity will often recur in the growing child from the persistent unequal muscle pull. For this reason, the question is raised as to whether, in the young and adolescent children who will require fusion, it would not be wise intentionally to attempt some type of muscle sacrifice to balance the muscle pull.

Some mention may not be amiss regarding the anaesthesia employed with these patients who are so often poor surgical risks. The usual anaesthetic used is gas and oxygen or cyclopropane, but in a few patients presenting marked reduction of vital capacity a regional block, using one-half-per-cent. novocain, has been done. The authors have been much

pleased with the uneventful, easy convalescence following this anaesthetic. Coincident with the anaesthesia consideration, an adequate fluid balance is most necessary. This has been achieved by the preoperative, operative, and postoperative administration of saline, glucose, or whole blood. By careful attention to the patient's general condition, the risk of operation upon the badly deformed and paralyzed patient has been reduced to a minimum. In fifty-eight fusions done within the last two years by the authors there has been no mortality.

SUMMARY

Poliomyelitic involvement of the trunk muscles is a serious condition, and the patient may be completely incapacitated by the resulting severe deformities.

A scoliosis has developed in 150, or 30 per cent. of 500 chronic poliomyelitis patients consecutively examined.

The most important factors for the development of the curves have been a lack of early absolute recumbency, the presence of a marked asymmetrical muscle paralysis, and a lack of adequate supportive treatment from a conservative and operative standpoint.

Twenty-one additional patients were found with symmetrical trunk paralysis, in whom scoliosis has not developed. It is the authors' contention that a progressive scoliosis will usually not develop in cases with balanced trunk paralysis.

Seventeen patients, or 3.4 per cent. of the total cases, had a complete recovery from their known paralysis.

Various types of operative and non-operative treatment, with certain modifications, have been found useful in the fusion operation for paralytic scoliosis.

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SPONTANEOUS FRACTURE OF THE FEMORAL NECK FOLLOWING IRRADIATION

Report of a Case

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In the use of higher voltage irradiation for the treatment of malignancies, it has been possible to increase the roentgen-ray dosage to the deeper tissues. This more efficient therapy has resulted in an increased danger of damage to the adjacent normal tissues, among which is the skeletal system.

In 1926 Ewing, in a report on tissue reaction to radiation, described what he termed "radiation osteitis". The following year Baensch of Leipzig reported the first case of spontaneous fracture of the femoral neck following roentgen therapy for carcinoma of the cervix. The American literature contained no reference to this subject until 1936, when Dalby, Jacox, and Miller brought to light fourteen instances of spontaneous fracture of the femoral neck following irradiation for cancer of the female generative tract. Their study was continued by Peck [8] who added fourteen original cases in a report of twenty-eight spontaneous fractures of the neck of the femur, seen in a group of 1,026 patients treated with irradiation to the pelvis for malignancies in the pelvis. Like Baensch's original case, five of the twenty-eight patients had bilateral fractures. Recently Batt and Hampton have reported such a fracture in a sixty-year-old white male, who had received radiation following amputation of the penis for adenocarcinoma.

PATHOLOGY

Pathological studies have been carried out in six of the reported cases [2, 4, 6, 8]. In each instance the findings in the region of the fracture have been essentially the same,—namely, (a) obliteration and sclerosis of the blood vessels, (b) narrowing and irregularity of the trabeculae with an increase in the fatty marrow intermingled with necrotic debris, (c) absorption of bone and widening of the marrow spaces, (d) lack of new bone formation, and (e) no evidence of metastasis at the site of the fracture. Okrainetz and Biller demonstrated roentgenographically an area of bone absorption in the upper portion of the head of the right femur of a patient who had received roentgen therapy for a papillary adenocarcinoma of the ovary. The change appeared thirteen months following the therapy and eleven months preceding a spontaneous fracture of the neck of the femur. According to Peck [7] the initial changes usually develop in the superior

portion of the femoral head close to the neck. This observation has been confirmed in the reported case.

SIGNS AND SYMPTOMS

Philipp saw five patients in whom fractures of the femoral neck occurred following roentgen therapy. In none of his cases was there a history of injury. Of the fourteen patients reported by Dalby, Jacox, and Miller, there was no instance of a history of trauma. In every case pain antedated the diagnosis of fracture. The pain was generally located in the hip and radiated into the anterior thigh and knee. An average of seven months elapsed from the onset of hip symptoms until the fracture was demonstrated.

TREATMENT

Too few cases have been seen for a standard of treatment to be established. It has been pointed out that the production of callus is unlikely, but occasionally fibrous union develops sufficiently to permit a partial return of function. The fact that in a number of the reported cases the head has maintained its apparent normal calcium content indicates that the use of internal fixation might give a fair percentage of good end results. Badgley, after observing a patient for a year or more, performed Colonna's reconstruction operation. Too short a time had elapsed for a result to be given. In the case here reported, "blind nailing", before the fracture was completed, was suggested but not urged.

CASE REPORT

V. S. (A 16731). On March 14, 1939, a fifty-nine-year-old female was seen with a complaint of vaginal bleeding of five months' duration. Biopsy of the cervix showed a squamous-cell carcinoma. Roentgen therapy was given daily for a period of three weeks, 3,000 roentgen units were given over four pelvic fields, two anterior and two posterior, each 10 by 15 centimeters. No irradiation was given over lateral or side portals. The following formula was used: 400 kilovolts, 5 milliamperes, 70 centimeters anode skin distance, 4 millimeter copper filter. She tolerated the roentgen therapy well, and on May 18, 1939, was given 4,500 milligram hours of intra-uterine and intracervical radium, and 2,500 milligram hours of vaginal application.

In November, 1939, she had some transitory pain and stiffness in the right hip.

In March, 1940, she again developed pain in the right hip. On examination there was some tenderness about the hip joint. Motion was limited and accompanied by muscle spasm. There was no shortening and the bony landmarks were in normal relationship. An anteroposterior roentgenogram showed an incomplete fracture through the upper cortex of the subcapital region of the right femur (Fig. 1). A lateral roentgenogram did not demonstrate the defect. It was thought that the patient had an osteonecrosis of the neck of the femur, or possibly miliary metastases without typical roentgenographic changes. She decided against immediate "blind nailing" or other therapy, so was instructed to remain in bed for a period of two weeks and to report back to the clinic for roentgenographic examination at the end of four weeks. After a social-service follow-up she returned on May 20, 1940, fourteen months following the roentgen therapy and six months following her first symptoms of pain in the right hip.

Examination showed typical findings suggestive of a fracture of the right femoral neck. Roentgenograms showed a complete fracture through the subcapital region with

FIG. 1

Anteroposterior roentgenogram of the right hip taken on March 29, 1940. Arrow points out defect of incomplete fracture. Lateral roentgenogram of the neck of the femur did not demonstrate the fracture.

FIG. 2

Anteroposterior roentgenogram of the right hip taken on May 20, 1940. There is a complete fracture through the subcapital region of the neck of the femur with some absorption at the site of the fracture and slight upward displacement of the shaft.

FIG. 3

Roentgenogram taken February 12, 1941. There is further rarefaction of the head and some sclerosis along the fracture site, but no signs of metastasis.

some absorption of the neck and upward displacement. There was rarefaction of the bone but no definite signs of metastases (Fig. 2).

She was having very little pain, was getting about with one crutch, so decided against any form of therapy with the understanding that one of the reconstruction procedures could be carried out later if necessary and advisable.

The patient has not returned for further examination. On February 12, 1941, through the cooperation of a roentgenologist in the patient's home city, further roentgenograms were made. There was further rarefaction of the head and some sclerosis along the fracture site, but no signs of metastasis (Fig. 3).

CONCLUSION

1. With the advent of higher voltage irradiation there is an increased danger of damage to the deeper tissues in the irritated area.

2. Bone, as well as the soft parts, is subject to injury by the roentgen ray.

3. If radiation osteonecrosis involves the femoral neck, spontaneous fracture may possibly occur.

4. When there is pain about the hip joint following irradiation for gynecological cancer, skeletal metastases should not be assumed without definite roentgenographic evidence.

5. In no way is the deep roentgen-ray therapy condemned, nor is its efficacy in pelvic malignancies questioned. We should continue its use and at the same time be alert to its unavoidable complications.

Appreciation is expressed to the Roentgenographic Department for assistance in preparing this report.

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AN UNUSUAL CASE OF ACTINOMYCOSIS

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This communication is presented to add to the published results of actinomycotic lesions submitted to surgical treatment. It is hoped that this record may assist others in the decisions to be made in the management of such cases.

CASE HISTORY

On May 31, 1939, a male patient, aged seventeen, complained of a dull pain and swelling in his right upper back.

About two months before, he had noticed some swelling about the third molar tooth of the right lower jaw associated with considerable pain. According to the dental examination on March 29, 1939, inflammation was limited to the periodontal membrane and the tissues immediately surrounding the affected tooth. Roentgenograms taken at that time showed a vertically impacted mandibular third molar on the right side. The occlusal surface was free of osseous structure and the impaction was due to lack of space. After preliminary hot saline irrigations, the tooth was extracted under local anaesthesia on March 30, 1939. Several sutures were used to close the wound. The patient was kept in bed a few days with routine irrigation of the socket. Healing was uneventful and he was dismissed April 11, 1939.

A short time later, while taking a bath, he noted an unevenness in his back when he leaned against the tub, and was able to palpate a swelling in the vicinity of his right upper back between the scapulae. It was at first small, but later increased in size and became tender. This was relieved by hot bathing. At about the time he first noticed the swelling he fell on his right side doing gymnastics at school and thought he had injured his back. He did not tell his parents about the swelling until a few days previous to seeing the author when, after playing several games of tennis, he noticed that his right arm became very weak and he could not continue the game. A relevant point regarding personal habits was that the patient chewed grass and leaves frequently.

A physical examination on May 31, 1939, showed a spare young man of asthenic habitus with a temperature of 98.8, pulse 72, and respiration 18. There was a tender diffuse swelling deep in the muscles between the vertebral spinous processes and the vertebral border of the right scapula, which extended from the levels of the third to the sixth thoracic vertebral spines. The skin was not attached and fluctuation was present. The scapula moved over the swelling on bracing back the shoulder. There was slight local elevation of temperature, but no redness of the skin. No other abnormalities were found. The gum of the previously involved molar was normal.

A provisional diagnosis of cold abscess was made and the patient was immediately admitted to the Hospital where the author inserted a wide-bore needle and aspirated approximately ten cubic centimeters of thick pus. On the following morning a bacteriological report of the smear gave the diagnosis of actinomycosis.

Roentgenographic examination showed:

1. The bone in the region of the posterior portion of the right mandible was normal.
2. There was no evidence of any disease involving the spine. The lungs were clear, with the haziness over the right upper chest probably due to the soft-tissue swelling in the upper posterior portion of the right thorax. There was a questionable area of

beginning bone destruction involving the sixth posterior rib in the region of the scapular line.

The report from the blood examination was:

Red blood cells—4,520,000
Hemoglobin—90 per cent.
Color index—1.01
White blood cells—8,500

In view of the fact that this case was diagnosed and prepared for operation without the presence of secondary infection or of sinus formation, a discussion took place regarding the best procedure. All agreed to the principle that treatment should be a resection of the whole area involved, if that were possible. It was also agreed that the infection had entered by the third molar tooth on the right lower jaw and that a track must run from that area to the area of the abscess. A conflicting point was the roentgenographic report that the sixth rib was eroded. If this were so, it would seem impossible to do a resection of the area without either removing a rib or opening the base of the abscess. It was decided before operation that the abscess probably lay under the rhomboid group and the trapezius. The methods discussed by which the resection could be carried out were:

1. The abscess could be aspirated as completely as possible and lipiodol injected in order to study the extent of the abscess, as it was agreed that unless this were known, one could not be sure of what was being done at operation. This was ruled out as an impractical method.

2. The abscess could be opened at the beginning and its contents washed out, and then, after its extent had been studied, it could be resected. This would entail allowing the contents of the abscess to contaminate the wound. Some considered that this would not make a difference if it were later washed out.

3. Dr. Miller suggested that, if the abscess were resected widely, there was a small chance that it might not be opened, depending on whether or not it extended to the rib. The results in a previous case treated in the Hospital seemed to be against this method. However, it was decided to proceed on these lines, since, if the abscess were opened from the deep aspect, that opening could be used for exposing the extent of the abscess cavity.

FIRST OPERATION

On June 6, 1939, an incision, about twelve inches long, was made over the abscess, extending from the upper free border of the trapezius downwards mid-way between the scapula and the dorsal spines. This incision was made to include an elliptical area of skin, two inches wide at its greatest width, to be removed with the abscess. The skin was then dissected back on each side for about two inches. The free lower border of the trapezius was defined and sectioned vertically upwards in relation to the scapula, and the rhomboids, major and minor, were severed from their attachment to the scapula. In this way, dissection down to the deep fascia overlying the semispinalis group of muscles was accomplished. The dissection then proceeded from below laterally under the serratus posterior superior and the aponeurosis of the semispinalis group of muscles This aponeurosis was taken with the abscess as the latter was located directly upon it.

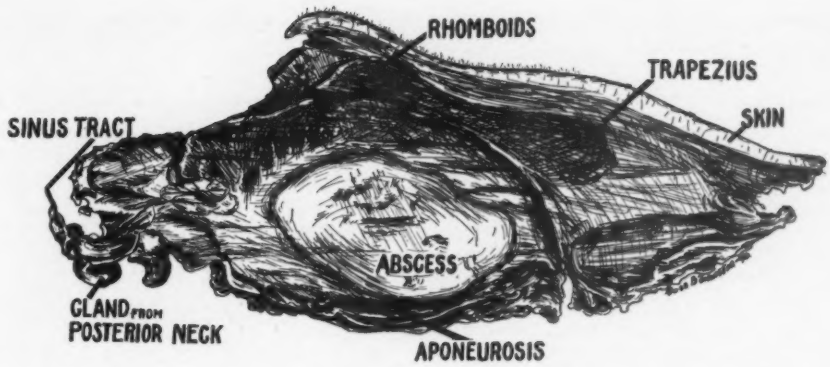

FIG. 1

Actinomycosis of back.

The dissection proceeded medially as far as the dorsal spines, which were cleared of the muscles and aponeurosis to the same depth. The whole mass was dissected upwards to the neck and into the lower part of the neck, under the levator scapulae muscle. At the deep part of the wound, a gland was seen which was removed with the mass and from its upper border, a cord, about the size of a piece of string and three inches in length, was removed. This was regarded as the track along which the infection had spread downwards to the gland and from there to the fascial plane under the trapezius and levator scapulae. The abscess was not opened at all during the operation and it was believed that the whole of the infected area was removed *en bloc*. The wound was irrigated with hydrogen peroxide followed by saline, and a few catgut stitches were used to hold the loose parts of muscle in place. It was considered before the operation that the whole wound should be left open as an ulcer to be later grafted, but in view of the clean dissection, the wound was sutured in its entirety with two small drains to evacuate any hemotoma.

Pathological Report

Macroscopic Examination. The specimen consisted of a piece of tissue measuring twenty by eight centimeters. One surface was covered with an elliptical piece of skin to which was attached subcutaneous tissue and muscle. On the deep surface there was a sinus tract eight inches long with a lymph gland the size of a marble at one extremity. Within the mass was a soft fluctuating area resembling an abscess. The specimen was fixed before opening.

Microscopic Examination. A large number of sections were taken through the tissue so as to give a topographical picture of the lesion. They showed an extensive inflammatory process which expressed itself as foci of purulent exudate, marked proliferation of fixed tissues, and fibrosis. The fibrotic area formed a wall separating the more active areas from the invaded muscle beyond. In the purulent areas the ray fungus could be identified.

The tract which appeared to extend up into the neck consisted of fibrous tissue, but no purulent area, and the gland which was sectioned separately showed catarrhal irritative changes with many germinal centers, but no histological evidence of the fungus.

Diagnosis. Exudative, purulent, proliferative, and productive inflammation of muscle due to actinomycosis.

SECOND OPERATION

On the twelfth postoperative day, the temperature, which for the previous two days had been normal, rose to between 99 and 100 degrees. The upper area of the wound at the level of the scapular spine became reddened and foul pus was discharged from the

small first stitch hole. The contents of the abscess were aspirated and examined microscopically. Some green negative actinobacilli and some fibrils were found. It was, therefore, decided to saucerize the wound immediately.

On June 20, 1939, the upper area of the wound was reopened to the extent of four inches, and an area of skin, two inches at its greatest width, removed. It was found that the abscess extended up to the area where the gland had been removed. This led to the belief that the infection had originated there. The wound was carefully cleansed, and packed with 20-per-cent. thymol in olive oil. The dressing was repeated daily.

Pathological Report

Macroscopic Examination. The specimen consisted of a piece of skin and subcutaneous tissue eight by one by one centimeters in size. In the center of the skin there appeared to be a partially healed incision which was greenish in color and appeared to be covered by a purulent, dense, solid exudate. The subcutaneous tissue oozed greenish pus on pressure. There was considerable dense, white, fibrous tissue in the subcutaneous tissue and little fat.

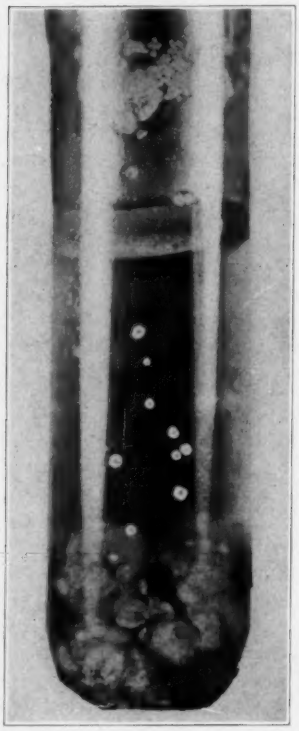

FIG. 2

Actinomyces grown in Robertson's meat. Note the rosettes adherent to the wall of the test tube.

Microscopic Examination. The section showed a skin surface with a V-shaped ulcerated area which extended down to the subcutaneous tissue. The ulcer had a necrotic base and was lined by inflammatory granulation tissue, more fibrotic in the deeper portion. No actinomycosis could be demonstrated histologically in this tissue.

THIRD OPERATION

Another abscess developed three inches below the first secondary abscess, so on June 27, 1939, the whole of the original scar around the sinus was removed, leaving a wound twelve by four inches to heal by granulation. The wound was packed with 20-per-cent. thymol in olive oil.

Pathological Report

Macroscopic Examination. The specimen consisted of a piece of skin and subcutaneous tissue measuring four by two by one centimeters.

Microscopic Examination. Sections showed an overlying skin surface beneath which, in the subcutaneous tissue, were numerous tracts filled with purulent exudate and proliferation of fixed tissues, and through which extended an inflammatory tract leading down to a large area of inflammatory granulation tissue in the subcutaneous regions. No actinomycosis could be identified in this histologically.

BACTERIOLOGICAL INVESTIGATION *

The material received at the laboratory consisted of five cubic centimeters of thick, dirty, yellowish-colored pus. Careful macroscopic examination showed numerous small pale-yellow granules. On fresh microscopic preparations of formol saline, lactophenol, and lactophenol with Amann blue, the characteristic granules of an actinomycotic infection could be seen. Each granule was formed by many rosettes consisting of a central mass of mycelium surrounded by clubs of different sizes. Smears

* The bacteriological investigation was made by L. P. Demers, M.D., and M. F. Howie, M.D.

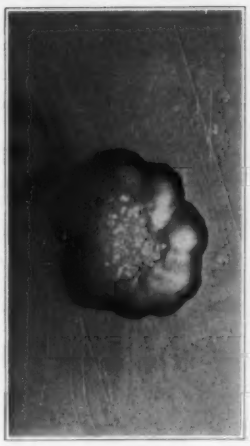

FIG. 3

Actinomyces grown on blood agar. Colony of Type 1 (× 10).

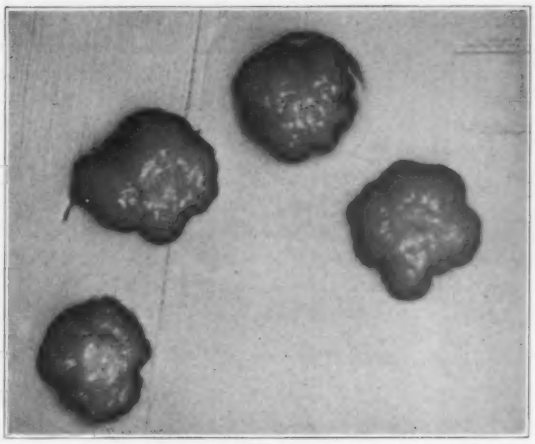

FIG. 4

Actinomyces grown on blood agar. Colony of Type 2 (× 10).

of the pus stained by Gram's method showed large numbers of pus cells; large numbers of irregularly stained, twisted, S-shaped or spiral gram-positive filaments which varied in length and width; long, slender, gram-negative filaments; and small gram-negative coccobacilli. The aerobic culture yielded no growth. The anaerobic culture after two weeks' incubation in a Fildes-McIntosh jar yielded:

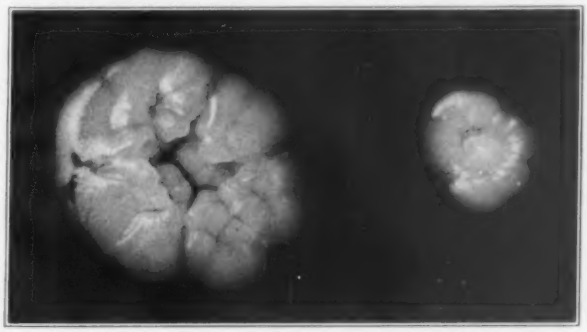

FIG. 5

Actinomyces grown on plain agar after two weeks of incubation. Colony of Type 2 (× 10).

I. Two types of actinomyces of the Wolff-Israel type.
II. One type of anaerobic gram-negative filament.
III. One type of anaerobic gram-negative coccobacillus.
IV. One type of anaerobic gram-positive coccus.

All these were obligate anaerobes.

I. Actinomyces of the Wolff-Israel Type

On blood agar these actinomyces developed two types of colonies:

Type 1 was rather soft, and not adherent to the substratum. The colony of this type had the form of a rosette with the central part raised and moruloid. The peripheral part was divided into lobes by several grooves radiating from the center to the periphery like the spokes of a wheel.

Type 2 was very hard to crush, very adherent to the substratum, and partly embedded in the medium. The colony of Type 2 also had the form of a rosette, but was without any definite pattern (mammillated).

These actinomyces grew well in liquid media, with the exception of tryptone water. The liquid remained clear, and rosettes formed and collected at the bottom of the test

tube or adhered to the wall. These actinomyces also grew well on ordinary solid media, such as plain agar, blood agar, Sabouraud's agar, glucose tartaric acid, corn syrup, plain potato and glycerine potato; but they failed to grow on Chandelier's or McConkey's agar. They did not form spores or aerial hyphae, did not produce true pigment although they developed a pale yellow color, or liquefy gelatin in which they grew fairly well, or produce any hydrogen sulphide (H_2S). They acidified milk without the formation of a clot, and fermented the following Hiss sera sugars (with acid reaction and clotting of serum): lactose, salicin, dextrose, maltose, raffinose, levulose, and starch, but did not ferment mannite, glycerine, inulin, sorbite, or galactose. Gram's smears from the cultures showed that the mycelia lose their morphology and resemble diphtheroids. Granules treated by absolute alcohol for a few minutes, or by potassium hydroxide (KOH) failed to grow on Sabouraud's agar or in Robertson's meat.

II. Gram-Negative Filaments

On blood plates these organisms formed at first small rosettes resembling a minute daisy, but, as they continued to grow, formed a large pin-head colony with effuse arborescent edges, which grew in the depth of the medium. The Gram's smear showed long slender gram-negative filaments, which grew well in the usual liquid media with the exception of tryptone water. In pneumobroth there was formation of paillettes which adhered to the walls of the test tube or collected at the bottom. They also grew on ordinary solid media, such as blood agar, or plain agar. They did not show any visible growth on corn syrup, glucose tartaric acid, Sabouraud's agar, plain or glycerine potato, or Chandelier's or McConkey's agar. They grew in gelatin without liquefaction. Their reaction in lead acetate was negative; in litmus milk, neutral. There was no reaction in Hiss sera.

III. Gram-Negative Coccobacillus

This gram-negative coccobacillus grew on blood agar as a small pin-head, slightly raised, semiopaque colony. It grew poorly in usual liquid media, and failed to grow in tryptone water. It also grew poorly on blood agar, and plain agar, and failed to grow on Chandelier's or McConkey's agar, glucose tartaric acid, or corn syrup. Its reaction in litmus milk was slightly acid, but it formed no clot. It grew in gelatin without liquefaction. Its reaction in lead acetate was strongly positive. There was no reaction in Hiss sera.

IV. Gram-Positive Coccus

This gram-positive coccus grew on blood agar as a small, conical, semiopaque colony with faint beta hemolysis. It grew well in ordinary liquid media, such as glucose broth and peptone broth, but failed to grow in tryptone water. It grew on ordinary solid media, such as blood agar and plain agar, but failed to grow on Chandelier's or McConkey's agar. It fermented the following Hiss serum sugars: lactose, dextrose, saccharose, maltose, and starch, but did not ferment mannite, salicin, raffinose, inulin, levulose, glycerine, galactose, or sorbite. Its reaction in lead acetate was negative; in litmus milk, acid, but without clot. It grew in gelatin without liquefaction.

FURTHER TREATMENT

General Treatment. This was carried out as for tuberculosis with stimulating diet and general exposure of the body to sunlight and fresh air.

Specific Treatment. Iodides were begun on June 7, 1939, and gradually increased to a maximum of 300 minims of Lugol's iodine a day, which was reached on July 8, 1939. They were gradually diminished until cessation July 14, 1939. The iodides caused considerable digestive disturbance. A course of vaccine therapy was given by Dr. Hugh Starkey beginning July 15, 1939, and continuing with weekly injections for six weeks.

Local Treatment. After the third operation the wound was dressed with 20-per-cent.

thymol in olive oil, and exposed to direct sunlight for increasing periods. Later these dressings were alternated with alphamel. On July 16, 1939, the patient was discharged to the country where the treatment was continued. For three weeks he was at the seaside where the wound was exposed both to sunlight and sea water. His general and local condition improved rapidly and on October 10, 1939, the wound was completely healed.

At that time graduated exercises of the muscles of the shoulder region were begun and resistance exercises were also given to increase their tone.

On May 15, 1940, the wound was sound. The patient was playing tennis, and had skied and played hockey during the latter winter months. Full movements were possible in his shoulder with little winging of the scapula or depression of the prominence of the shoulder. (See Figures 6-A and 6-B.)

Since then he has been readmitted for excision of the scar. The area was covered by Thiersch and pinch grafts, and on September 14, 1940, was completely healed. There has been no recurrence.

DISCUSSION

After the first operation the question arose as to whether the best method was used and whether it would be possible on another occasion to carry it out in a more advantageous manner. As a result of the operation, the whole of the trapezius muscle attached to the scapula (but not the clavicular head) with the rhomboids, major and minor, were removed. The serratus anterior was left as was the levator scapulae and it was a question as to what stability the scapula would have in the movements of the shoulder. Theoretically speaking, the whole of the trapezius could have been divided and resutured back in place, had the extent of the abscess been known. This would have given better functioning of the scapula.

Further, the primary suture of the wound might be criticized in the light of the subsequent course. The author feels it achieved a most

FIG. 6-A FIG. 6-B

Photographs showing range of motion fourteen months after third operation.

important result,—that is, the complete sealing of the large space under the scapula and serratus anterior in continuity with the wound of the operation when the trapezius and rhomboids were removed.

This case is unusual in several ways. In the first place, the location of the abscess is uncommon. No case with the abscess in this location is mentioned in Cope's monograph. Further, the history and dissection correlated and traced the entry of the organism very definitely to the third molar tooth. Again, it is unusual for the diagnosis to be established before secondary infection has been introduced. Finally, it is unusual in such a situation to be able to resect the whole, or practically the whole area involved. The author is also amazed to find the splendid functional result in the shoulder considering the extensive removal of muscles. It is hoped that this is a cure. Should a recurrence occur, it will be reported.

The author wishes to acknowledge the valuable help given him in this case by C. J. Tidmarsh, M.D., and Gavin Miller, M.D.

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OSTEOCHONDROMATOSIS

BY CLIFFORD LEE WILMOTH, M.D., F.A.C.S., PITTSBURGH, PENNSYLVANIA

Osteochondromatosis is a relatively uncommon pathological condition in which multiple intra-articular cartilaginous bodies form. These live in the joint cavity either as attached or free bodies. The etiology of this condition continues to be in doubt, and the part which infection, trauma, embryonic rests, and neoplastic changes play in their production is a matter of considerable difference of opinion. This report covers a ten-year period starting in 1930. During this period nine patients with osteochondromatosis have been seen, six with involvement of the knee joint, two of the elbow, and one of the hip. The patients were all males varying in age from twenty-six to sixty-four years.

CASE 1. A male, aged sixty-four years, for eight years had experienced pain in the left knee joint, and for six years had been aware of a hard nodular mass in the popliteal region. He had quite a pronounced limp on walking. Examination showed a marked instability of the left knee joint, a mass of hard irregular bodies which could be palpated in the popliteal area, and a quite marked atrophy of the muscles of the left thigh and calf. The roentgenogram showed the popliteal mass to be made up of multiple cartilaginous bodies (Fig. 1-A). Through an anteromedial incision all loose bodies were removed

FIG. 1-A

Case 1. Roentgenogram shows osteochondromatosis of the knee joint in the nonactive stage. The bodies are almost all in the posterior pouch, and many were found deeply buried in the soft tissues of the popliteal area.

FIG. 1-B

Case 1. Photograph of the loose bodies removed from the knee joint.

(Fig. 1-B). No attempt was made to remove those bodies which had penetrated into the tissues outside the synovial membrane and which were well embedded there. The patient was followed for a period of slightly over two years. When seen at the end of ten months, there was still marked instability of the left knee joint with associated muscle atrophy. The pain, however, had been largely removed by the operative procedure. When seen again slightly more than two years postoperatively, the clinical picture was unchanged. The roentgenogram failed to show the formation of any new loose bodies, and the ones which were embedded in the tissues of the popliteal area had not changed in size or position.

CASE 2. A male, aged forty-six years, came to the hospital because of pain in the left hip of two years' duration. Examination showed slight limitation of motion as compared to the right hip. There was very slight atrophy of the thigh muscles. The diagnosis was made by roentgenogram which showed the joint capsule filled with osteocartilaginous shadows (Fig. 2). Through a Smith-Petersen incision the hip joint was opened and the small loose bodies removed. The patient was seen only once, eight months after leaving the hospital. There was no pain and no limitation of motion nor any appreciable atrophy of the thigh muscles. The roentgenogram failed to show any recurrence of osteocartilaginous bodies.

FIG. 2

Case 2. Roentgenogram shows osteochondromatosis of the hip joint.

CASE 3. The patient was a male, aged thirty-two years, who for a year had noticed occasional twinges of pain in the right elbow. Physical examination was essentially negative for any objective pathology. The elbow joint showed no tenderness or swelling, nor was there any atrophy present. Motion was not limited. The roentgenogram showed osteocartilaginous bodies of small size in both the olecranon fossa and the anterior joint space. Bilateral incisions were made and all the loose bodies found were removed. All the synovial tissue which could be reached was excised, especially that about the epicondyles at the synovial-cartilaginous junction. Recovery was uneventful and the patient when last seen was free from pain al-

though the postoperative roentgenogram had shown a few minute bodies which had been missed at the time of operation. This patient could not be located for later follow-up records.

CASE 4. The patient was a male, aged thirty-six years, who complained of pain and slight swelling of the left elbow joint for a period of four years. The motion of the joint was becoming less, and he had been aware of a firm hard mass in the anterior aspect of the joint for a year. Upon examination there was limited flexion, extension, and rotation of the left elbow joint, and moderate atrophy of the muscles of the left forearm. The grasp of the left hand was decreased. The roentgenogram showed multiple osteocartilaginous bodies filling the anterior joint space and the olecranon fossa (Figs. 3-A and 3-B). Through bilateral incisions into the elbow joint the loose bodies (Fig. 3-C) were removed and a partial synovectomy was done. That part of the synovial membrane which had attached cartilaginous bodies, or which showed definite changes, was removed. The patient was seen after eight months and again after three years. There was very slight residual loss of rotation of the radius, and there was a loss of 10 degrees in extension. A few small bodies missed at the time of operation showed no apparent change in size or number in roentgenograms after three years. Subjectively the results were quite satisfactory.

CASE 5. A farmer, aged fifty-two years, because of rheumatism in the right knee, had had a roentgenogram taken by his family doctor five years previously. It was found then that the knee joint contained many osteocartilaginous bodies. Roentgenograms taken at the time he was first seen by the writer failed to show any apparent alteration in the number or size of the bodies when compared with the roentgenogram taken five years before. The cartilaginous bodies were largely in the posterior compartment, with a few scattered loose bodies in the suprapatellar space. He stated that his symptoms had remained unchanged, and that he was able to do his farm work with only occasional discomfort. Because of the loose bodies in the anterior compartment, he was advised to have the knee operated upon, but this he declined to do. While no further change in size or number of the loose bodies was expected, it was believed that damage to the joint surfaces would occur if the cartilaginous bodies were not removed. This patient, replying to a follow-up letter three years after the last examination, stated that he had not noticed any appreciable change in the knee since the previous examination.

CASE 6. A salesman in a department store, aged fifty-eight, had experienced occasional periods of pain and swelling of the left knee over a period of eight years. The swelling of the knee joint followed sudden severe attacks of pain in the joint, and gradually subsided with rest and heat. The interval between attacks had been variable, but they were becoming more frequent, with more discomfort present between the attacks of severe pain and swelling. Examination showed slight instability of the knee joint. The patient walked with a limp and with the aid of a cane. There was moderate atrophy of the calf and thigh muscles. In the suprapatellar space a large loose body could be palpated. The roentgenogram showed multiple osteocartilaginous bodies. An anteromedial incision was made, and the loose bodies were removed. The synovial membrane adjacent to the articular cartilage of the femur was reddened and thickened, particularly over excrescences of soft cartilaginous material, and this was excised. This patient was seen at frequent intervals over the following four years. At the end of that time he still walked with a slight limp, and complained of some aching pain particularly in damp weather. His symptoms were those usually complained of in connection with arthritic changes. A recent roentgenogram shows definite arthritic changes, but no recurrence of osteocartilaginous bodies.

CASE 7. This patient was a male, aged twenty-six, who for two years had experienced painful swellings of the right knee joint. The examination was essentially negative except for the palpation in the suprapatellar space, of a freely movable hard body. The roentgenogram showed multiple osteocartilaginous bodies which were removed through

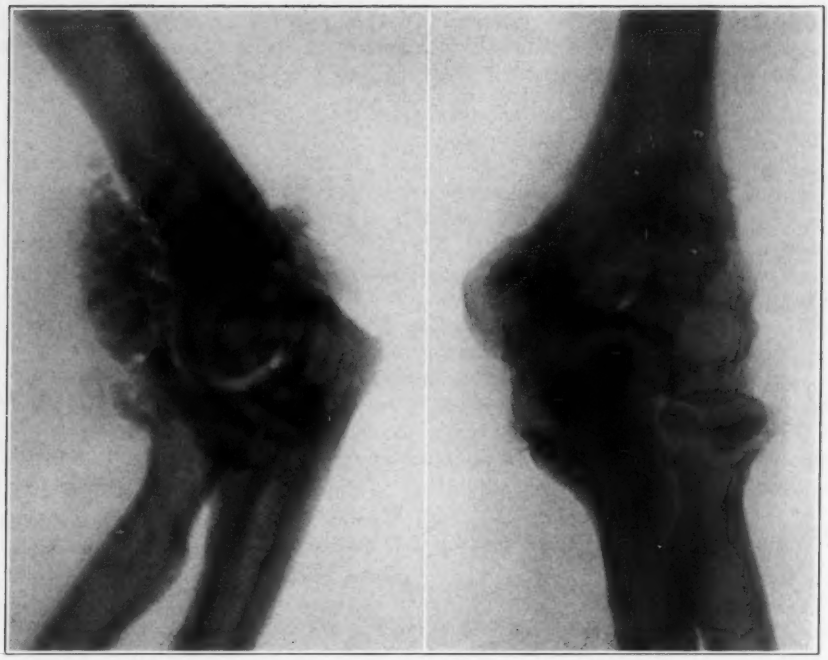

FIG. 3-A FIG. 3-B

Case 4. Lateral and anteroposterior roentgenograms showing osteochondromatosis of the left elbow joint. The bodies are largely in the anterior compartment and in the olecranon fossa.

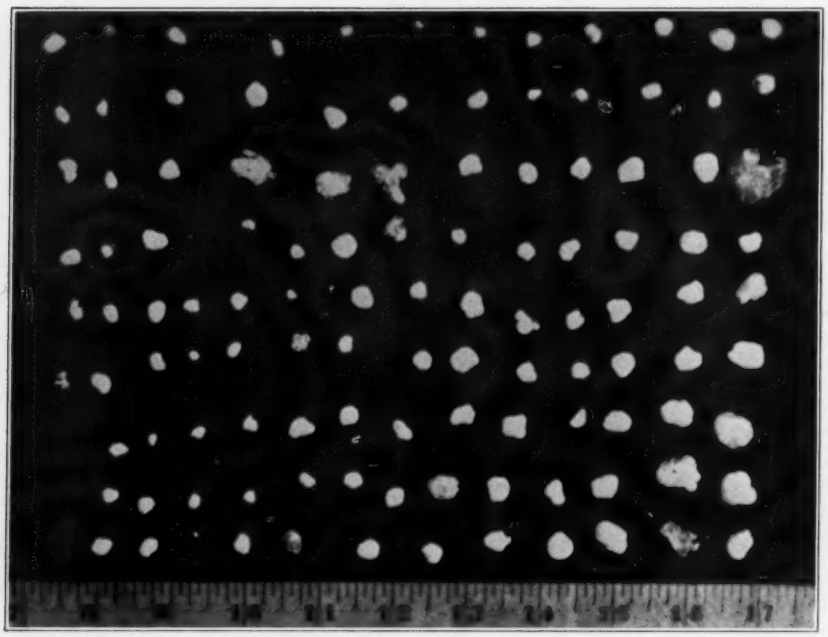

FIG. 3-C

Case 4. Photograph of the loose bodies removed from the elbow joint.

an anteromedial incision into the knee joint. The synovial membrane at the synovia-cartilaginous junction was reddened and thickened, and was excised. Soft excrescences of cartilaginous material were removed with a curette. This patient has been seen at intervals over the past three years. Pain of some arthritic type has been experienced occasionally. There is no limitation of motion and no demonstrable muscle atrophy. Roentgenograms show no recurrence of loose cartilaginous bodies. The embedded bodies in the popliteal area, which were not removed at the time of operation, have remained unchanged in size and location.

CASE 8. This patient was forty-two years old. He complained of rheumatic pains in both knees of three years' duration. Physical examination was essentially negative. Roentgenographic examination of both knees showed multiple osteocartilaginous bodies about the superior aspect of both patellae. An arthrotomy of each knee was done. The cartilaginous bodies were found attached to the synovial membrane bordering the patellae. The bodies, together with the synovial membrane, were removed. This patient was seen six and seventeen months postoperatively. On each occasion the roentgenogram showed no new bodies forming. There was no objective evidence of pathology present. He stated that he still had occasional arthritic pains in the knee joints.

CASE 9. The patient was a man of forty-five years. He complained of occasional pain and discomfort in the left knee joint, which had been present at intervals for a period of five years. Slight swelling of the joint occasionally accompanied the attacks of pain. Physical examination was essentially negative. There was no muscle atrophy, limitation of motion, or sign of effusion. An anteromedial incision was made into the joint cavity, and the loose bodies were removed. An area five centimeters in length across the femur was apparently the source of the loose bodies. The synovial membrane in this area was bluish red, and there was a piling up of soft cartilaginous material. A partial synovectomy was done. This patient could not be followed postoperatively.

The difficulties of a satisfactory follow-up system are apparent to all who attempt it. These patients were for the most part industrial workers who, because of the economic conditions of the last ten years, seldom remained long in one location. Even follow-up letters failed to reach them or were of little help. In changes as slowly progressive as benign osteocartilaginous growths, it would be much more interesting and helpful if a careful check could be made over a period of at least ten years. The longest period of this series of cases was four years.

The few cases that have been studied have shown two characteristics,—the distribution of the cartilaginous bodies in the joint cavity has been fairly constant, and localized synovial osteocartilaginous proliferation has been found at the synovial cartilaginous junction. The knee joint may be taken as an example. Most loose bodies were found in the posterior pouch, a few in the suprapatellar pouch, and an occasional body in other recesses of the joint cavity. Apparently most of the cartilaginous bodies gravitate into the more dependent portion of the joint which is the posterior pouch. As the bodies increase in size, they produce distention of the normal joint cavity which often progresses to sacculations or diverticuli from the main joint cavity. As Rixford[1] has stated, "It is as though the cartilaginous masses backed out through areas of least resistance carrying the expanded synovial membrane before them".

The appearance of the synovial membrane differed widely. When the cartilaginous bodies were in the posterior pouch of the knee joint,

FIG. 4

High-power photomicrograph shows the superficial zone of one of the small bodies in the active stage. The outer layer is a very cellular area of fibrous tissue,—the perichondrium. The deeper structure is fibrocartilage.

FIG. 5

Photomicrograph of a mature osteocartilaginous body. The superficial layer is formed of dense fibrous tissue,—the perichondrium. Well-formed fibrocartilage exists beneath this, and areas of lime-salt deposits are seen in the osseous area.

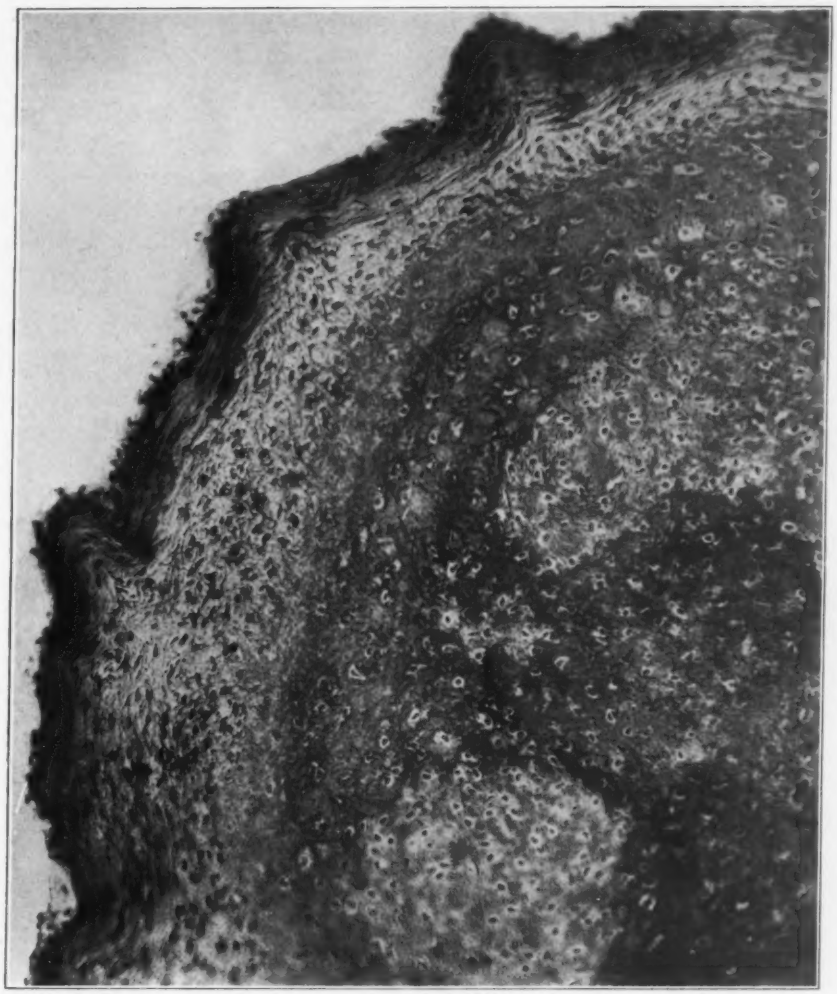

FIG. 6

Photomicrograph of an old non-active loose body which has ceased to grow. The superficial layer of fibrous perichondrium is thrown into folds and there is some exfoliation of the cells on the surface. The center of the body is made up of cartilage and bone. As calcification of the cartilage cells occurs, they appear more round and stand out distinctly because of the calcium deposit around the cell. In general it may be stated that the histological examination reveals a fibrous proliferated layer which passes over into hyaline cartilage, which in turn changes to calcified cartilage. Lamellae of bone which form are free from haversian canals and bone marrow.

the synovial membrane in the suprapatellar region showed little change or at most only a congestion and a slight thickening. Where the cartilaginous bodies were scattered throughout the joint cavity, the entire synovial membrane was thickened and irregular in contour.

The relative absence of subjective symptoms, compared to the marked internal derangement of the joint, was striking. There was seldom a demonstrable increase in joint fluid, and no local heat or redness

of the joint tissues. The size and number of the loose bodies was the chief factor in giving objective evidence of joint pathology. Several patients with knee-joint involvement walked with a limp. Except for occasional sharp pains, the general subjective symptoms were those usually complained of in arthritic joints. In four of the patients with knee-joint involvement, the cartilaginous bodies could be palpated either in the suprapatellar space or in the popliteal area. The diagnosis was confirmed in all cases by roentgenograms which gave a picture of multiple loose or attached osteocartilaginous bodies in the absence of, or with relatively little, other joint pathology.

Surgical removal of the osteocartilaginous bodies was advised in all instances. The difficulty of removal of all the bodies in the knee and elbow regions is apparent from postoperative roentgenograms. No effort was made to remove the cartilaginous bodies which had made their way through the synovial membrane of the posterior pouch of the knee joint. It was believed that these encapsulated bodies would cause no symptoms, would not change in size or position, and that their removal would not be without danger to important structures which pass through that region. An effort was made to remove every loose body or those which might become loose. It was believed that this was necessary if further joint damage was to be avoided. A partial synovectomy was done whenever the synovial membrane showed definite changes. Whether removal of the synovial membrane and the osteocartilaginous bodies will prevent a recurrence of the condition is unknown. In the few cases which were followed there was no recurrence of the formation of the osteocartilaginous bodies.

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COMPLETE BILATERAL EPIPHYSEAL SEPARATION OF THE UPPER HUMERAL EPIPHYSES DUE TO SCURVY

Report of a Case

BY RICHARD T. HUDSON, B.S., M.D., F.A.C.S., DENMAN C. HUCHERSON, A.B., M.D., AND ALVIN B. ORTNER, A.B., M.D., LOUISVILLE, KENTUCKY

Cases of scurvy with minor epiphyseal changes and subperiosteal hemorrhage are not rare, but the complete separation of both upper humeral epiphyses is unusual.

Skeletal changes of this magnitude result only from scurvy of severe degree, and after many weeks of avitaminosis.

In a review of the literature since 1917, the authors have not been able to find a similar case, with such extensive osseous changes.

CASE REPORT

W. L. D., a nine-months-old, white female, was admitted to the Children's Free Hospital on April 12, 1940. According to the parents the child was very nervous and resented handling because of apparent generalized tenderness. The family history was essentially negative; the patient was the youngest of ten children, the other nine living and well. The child had not been able to sit alone. Her diet since birth had consisted of one small can of Pet milk daily, diluted with water and Karo, and given in divided feedings. She had had no orange juice, cod-liver oil, cereals, or vegetables in her diet.

Three months prior to admission, the mother noticed that the right ankle was swollen and tender. This was followed by swelling and tenderness of the right knee and later the left lower leg, and then generalized tenderness on handling the patient. Three weeks after the onset the patient developed an up-

Fig. 1

April 15, 1940

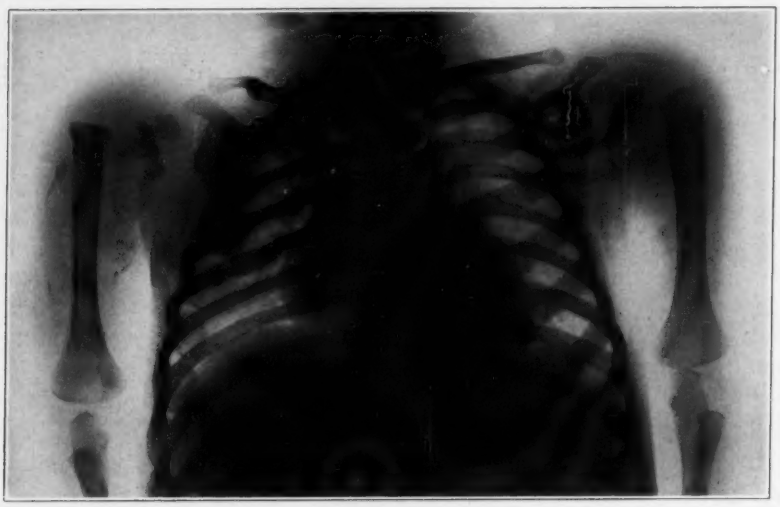

FIG. 2
April 15, 1940

FIG. 3
April 25, 1940

per respiratory infection, and was treated by the family physician, who prescribed cod-liver oil, which was soon discontinued because the child vomited. One month previous to admission to the hospital the lower extremities had become more swollen, and there was bilateral swelling and enlargement of the shoulders. These enlargements gradually increased in size, and the child failed to gain weight.

Physical examination revealed a poorly developed and undernourished, white, female child, nine months of age. The skin was pallid, dry, and wrinkled. The anterior cervical and inguinal lymph glands were palpable. The anterior fontanel was open one and one-half finger-breadths, and the posterior fontanel was closed. The thorax was flattened in its anteroposterior diameter, and a marked rachitic rosary was present. The heart and lungs were normal; the abdomen slightly distended; and the extremities, thin and underdeveloped, showed a rather marked generalized hypotonicity. There was little voluntary motion, and passive motion of the extremities caused the child to cry. An abnormal enlargement of both deltoid regions was very noticeable. The right knee was larger than the left, and there was a fusiform swelling of the left thigh. The reflexes were normal.

The laboratory findings were as follows:

Red blood cells—2,180,000
Hemoglobin—5 grams
White blood cells—21,500
Polymorphonuclear neutrophiles—31 per cent.
Small lymphocytes—68 per cent.
Eosinophiles—1 per cent.

There was a marked anisocytosis and poikilocytosis, and three normoblasts per 100 white blood cells were seen in the differential.

Urinalysis was negative.

A provisional diagnosis of advanced scurvy was made.

Roentgenograms of the entire body were taken on admission to the hospital. These showed wide separation of the upper humeral epiphyses and calcified subperiosteal hemorrhage about the humeri, femora, tibiae, and fibulae. There was an old healed fracture of the right clavicle. The epiphyseal lines of the bones of the lower extremities were widened and irregular, showing a definite *Trümmerfeld*.

On admission to the hospital the patient was given small blood transfusions, and vitamin-C therapy,—250 milligrams in twenty-four hours.

On April 19, 1940, the blood picture was as follows:

Red blood cells—4,260,000
Hemoglobin—13 grams
White blood cells—5,800
Polymorphonuclear neutrophiles—72 per cent.
Small lymphocytes—27 per cent.
Eosinophiles—1 per cent.

FIG. 4
May 23, 1940

The epiphyseal separations of the humeri were treated by manipulation under general anaesthesia, and the application of a double body spica with the arms in wide abduction.

Anteroposterior x-rays made on April 25, 1940, with the child in the cast (Fig. 3) showed fairly satisfactory reduction of the epiphyses of the humeri.

The child continued to improve, took food satisfactorily, and lost all tenderness on

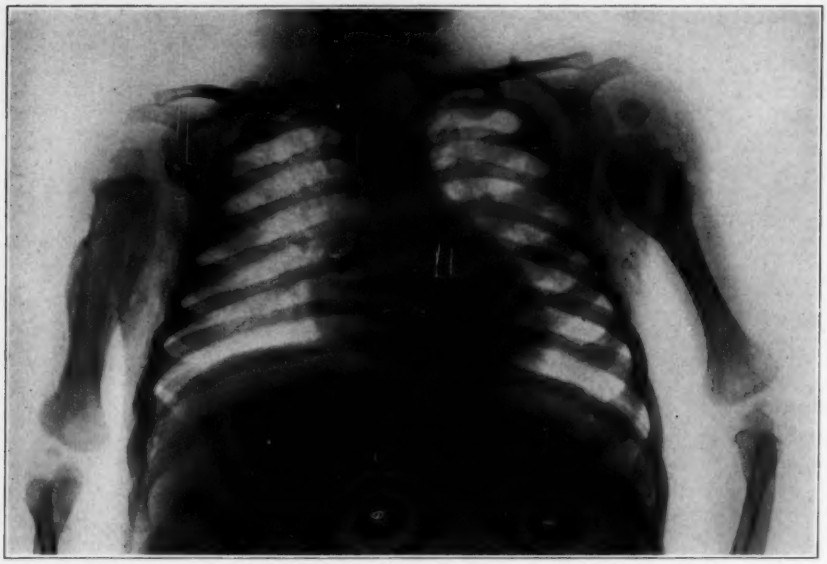

FIG. 5
June 4, 1940

handling. The cast was removed on June 4, 1940. Anteroposterior x-rays (Fig. 5) at this time showed the capital epiphyses to be in practically normal relationship with the shafts of the humeri. A marked persistent reaction and thickening about the bone ends were still present.

On her discharge from the hospital the family physician was advised to continue vitamin-C therapy.

On September 10, 1940, the physician reported verbally: "All the symptoms have disappeared, and the patient seems to be in excellent health."

LATERAL DISLOCATION OF THE HEAD OF THE RADIUS WITH FRACTURE OF THE ULNA

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From the Fracture Service of the Knickerbocker Hospital, New York

Dislocation of the head of the radius, occurring as an associated lesion with a fracture of the shaft of the ulna, is often overlooked. The frequent coexistence of these two lesions, emphasized by Monteggia, makes it imperative to examine carefully the relationship of the radius with the capitulum in all fractures of the shaft of the ulna. The radial dislocation is usually anterior, but may be posterior or lateral; the ulnar fracture is commonly in the upper third, but may be in the mid or lower third of the shaft, or through the olecranon.

The following case report illustrates a lateral dislocation of the head of the radius associated with an incomplete fracture of the proximal end of the ulna.

CASE REPORT

M. K., a boy of seven, was admitted to Knickerbocker Hospital on November 6, 1940, complaining of a painful, swollen left elbow. He stated that, while sliding down an incline, he tripped and fell, striking his left elbow against a concrete step.

Examination revealed a tender, markedly swollen left elbow. The forearm was pronated and held at a right angle with the arm. No active flexion, extension, or supination at the elbow was possible. The head of the radius was abnormally prominent on the lateral aspect of the forearm and exquisitely tender. There was no injury to the ulnar, median, or radial nerves.

Roentgenograms (Figs. 1-A and 1-B) showed an incomplete fracture of the proximal end of the ulna with lateral angulation, and a complete lateral, and slight anterior dislocation of the head of the radius.

After careful skin preparation, open reduction was performed on November 13, 1940, through a posterolateral incision as described by Boyd. A blood-pressure cuff tourniquet was used, and Lane technique was observed throughout. The incision began above the lateral border of the olecranon and extended downward along the shaft of the ulna. The insertion of the anconeus and the origin of the supinator were reflected from the ulna subperiosteally, and the muscles retracted radially, exposing the upper third of the shaft of the ulna, and the head and neck of the radius.

Pathology. The elbow joint contained 10 cubic centimeters of liquid blood and several blood clots. The head of the radius was dislocated laterally, slightly forward, and was separated from the radial notch. There was a complete tear of the orbicular ligament at its insertion into the ulna, and the articular cartilage lining the radial notch was lacerated. The ulna was angulated radially and several incomplete longitudinal fracture lines were present (Fig. 2).

Procedure. The angulation of the ulna was corrected by direct light blows through a blunt instrument placed at the apex of the angulation. The dislocation of the radius was reduced by traction and supination of the forearm, and the torn edges of the orbicular ligament were approximated with mattress sutures of silk. Fine interrupted silk sutures were used to close the capsule, the reflected muscles, fascia, and skin. The elbow was placed at a right angle in a posterior molded plaster splint.

FIG. 1-A

FIG. 1-B

Roentgenograms, before reduction, showing the lateral dislocation of the radial head with the incomplete fracture of the ulna. Note the slight anterior displacement of the radial head.

After-Care. The wound healed *per primam* and the patient was discharged on the seventh postoperative day. The splint was removed twenty days after operation, the arm placed in a sling, and active motion at the elbow encouraged. All motions at the elbow joint returned rapidly, and nine weeks after injury there was normal range of flexion, extension, pronation, and supination (Figs. 3-A and 3-B).

This case illustrates an unusual Monteggia fracture, in which the fracture of the ulna is incomplete and the dislocation of the head of the

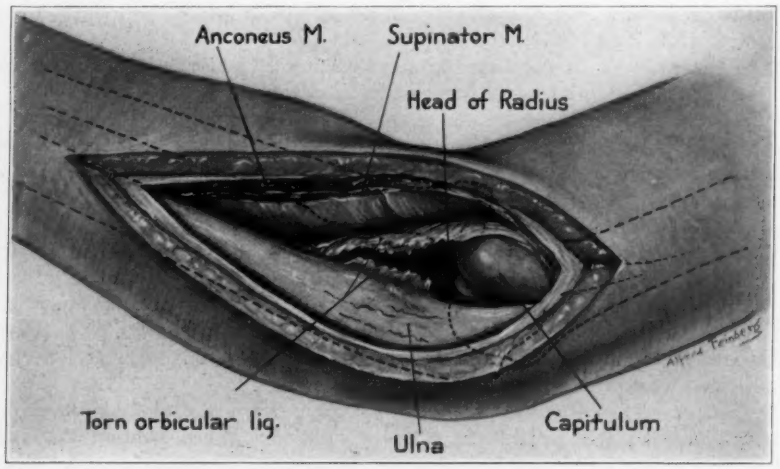

FIG. 2

Drawing made at operation. Note abnormal relationship of the radial head with the capitulum and the tear of the orbicular ligament.

radius is lateral. In Speed's report of sixty-two cases of Monteggia fracture the dislocation of the radial head was anterior in 83.3 per cent.

FIG. 3-A

FIG. 3-B

Roentgenograms after reduction. Note normal relationship of the radial head.

and lateral in only 6.7 per cent.

Boyd's incision is ideal for the operative treatment of patients with this injury, for it makes possible the exposure of the upper fourth of the radius and the shaft of the ulna through one incision, with complete protection for the deep branch of the radial nerve.

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ROENTGENOGRAPHIC STUDY OF THE CARPAL CANAL

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The palmar surface of the bones of the carpus is concave from side to side. This deep concavity is named the carpal groove and is formed by the shape and position of the bones at the sides of the carpus. Prominent bone structures on the ulnar border of the groove are the pisiform bone and the hook of the hamate or unciform bone. The radial side of the groove is formed by the tuberosity on the navicular or scaphoid bone and the thick projecting crest or ridge on the greater multangular bone or trapezium. Arched over the hollow of the wrist or carpal groove, and firmly attached to the bony boundaries, is the extremely strong transverse carpal or anterior annular ligament. The ligament converts the groove into the carpal canal or tunnel (Fig. 1). Through the carpal tunnel pass the median nerve, the flexor digitorum sublimis, flexor digitorum profundus, and flexor pollicis longus tendons. The borders of the canal provide origin or insertion for seven of the intrinsic muscles of the hand as well as insertion for the tendon of the flexor carpi ulnaris muscle.

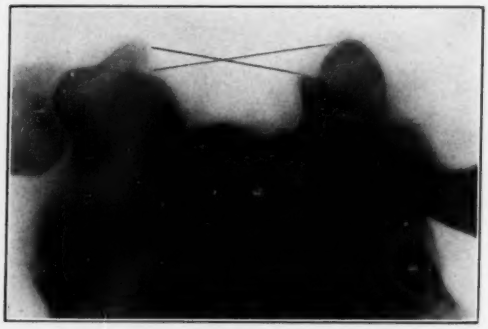

FIG. 1

Vertical roentgenogram of the wrist demonstrating (1) pisiform bone, (2) hook of the hamate or unciform, (3) tubercle of the navicular or scaphoid, and (4) crest of the greater multangular or trapezium. The crossed lines represent the transverse carpal ligament which converts the carpal groove into the carpal canal.

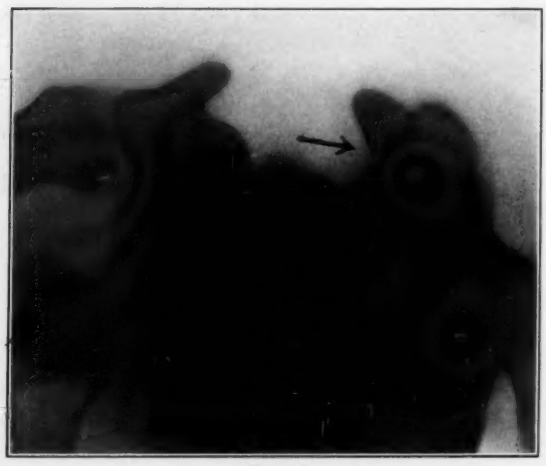

FIG. 2

Roentgenogram of the bony boundaries of the carpal canal which demonstrates a developmental defect or separate hook of the hamate.

The superficial branches of the ulnar artery and nerve cross the tip of the hook of the hamate, and the nerve can be felt slipping over the hook in the hypothenar eminence about two centimeters distal and

radial to the pisiform. The pisiform bone is easily palpated in the normal hand at the end of the flexor carpi ulnaris tendon. The tubercle of the scaphoid can be found at the point where the flexor carpi radialis tendon passes out of sight at the wrist. The crest of the trapezium is immediately distal to the scaphoid tubercle in the medial edge of the ball of the thumb; the two are often felt as one continuous bony prominence.

Very limited information is available regarding developmental anomalies (Fig. 2), injury (Fig. 3), or disease of the bony borders of the carpal canal. Additional knowledge would be gained if these anatomical structures were more frequently studied roentgenologically by the technique demonstrated in Figure 4.

FIG. 3

Roentgenogram of carpal canal demonstrating a fracture or a developmental defect of the hook of the hamate.

FIG. 4

Photograph demonstrates the technique for obtaining a vertical roentgenogram of the bony boundaries of the carpal canal. The flexor surfaces of the patient's forearms should rest comfortably upon the x-ray table and the cassette. The hand should not be in ulnar deviation in order to prevent superimposition of the shadows of the pisiform bone and the hook of the hamate. The position of the x-ray tube will vary according to the individual patient. The central ray should be parallel with and in front of the third metacarpal bone. The standard distance used is thirty inches from focal point of x-ray tube and cassette. X-rays should be made of both right and left carpal canals.

AN OSTEOPERIOSTEAL CHISEL

BY R. E. BURNS, M.D., MADISON, WISCONSIN

There has been much discussion in medical annals as to what constitutes the periosteum. There is, however, little debate regarding the rôle of the osteoperiosteal graft in the situation in which it is indicated. The purpose of this article is to describe the difficulties ordinarily encountered in the removal of the graft, with a suggestion for their solution.

The medial aspect of the tibia is the customary site for the removal of the graft. Here the periosteum is thick, and the cortex is thick and, too often, brittle. If a straight chisel is used in the removal of the graft, difficulty is often encountered. If the angle of inclination of the chisel is too small, the fibrous layer of the periosteum only is scraped off and very

FIG. 1

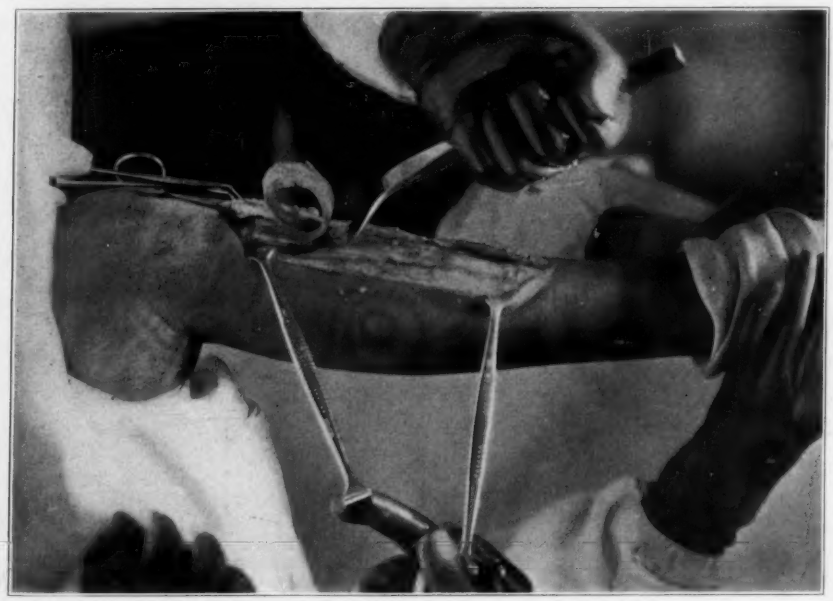

FIG. 2

little of the element of osteogenesis remains. If, on the other hand, the angle of inclination is too great, large pieces of the cortex together with the periosteum are removed. The graft as removed usually consists of an alternating series of loosely connected fragments of periosteum holding together a series of cortical grafts.

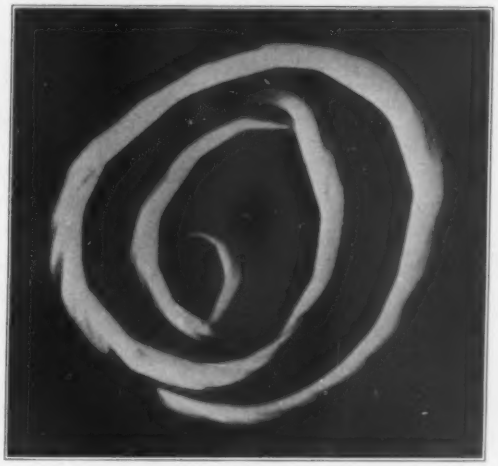

FIG. 3

It was thought that, if a chisel could be devised based on the principle of the plane gauge, it would ensure a graft of uniform thickness, which could be easily removed. The chisel is curved on the flat. Two guards prevent its penetration beyond three sixteenths of an inch. It can be driven abruptly into the bone until the guards are reached. Thereafter, due to the curve of the chisel, the hand of the operator in his removal of the graft parallels the bone. As the graft is removed it immediately curls up. This curling indicates the continuity of its substance. It can be molded to fit the desired location by cross hatching.

Figure 1 shows the chisel. Figure 2 shows the final step in the removal of the graft. Figure 3 shows a roentgenogram of the graft after removal.

TREATMENT OF FRACTURES OF THE NECK OF THE FEMUR BY INTERNAL FIXATION

REPORT OF THE FRACTURE COMMITTEE OF THE AMERICAN ACADEMY OF ORTHOPAEDIC SURGEONS *

INTRODUCTION

The first report of the Committee appointed to study treatment of fractures of the neck of the femur by internal fixation was rendered at the Annual Meeting of the American Academy of Orthopaedic Surgeons at Memphis, Tennessee, January 1939.

This was really not a committee report, as all the material had been collected and analyzed by Dr. H. Earle Conwell. Subsequent to the meeting, however, a committee was appointed, consisting of Dr. Willis C. Campbell, Dr. H. Earle Conwell, Dr. Lawson Thornton, Dr. Frank D. Dickson, and Dr. Herman C. Schumm. A most critical attitude was assumed by this committee. No case reports were accepted unless all data were complete. The result of this critical attitude was that the number of cases was reduced from 1485 to 241. A report based on this relatively small number of complete cases, with accurate information, is greatly to be preferred to a report based on a large number of cases with incomplete data.

This report represents the second installment of the study of internal fixation and the treatment of acute central fractures of the neck of the femur. It is planned to have the committee continue this study so that in another two years a third report will be rendered, based on further study of this same group of cases. This future report should be most instructive, particularly with reference to aseptic necrosis and hypertrophic changes.

The Academy is indeed grateful to the Chairman of the Committee and his coworkers for this outstanding, constructive piece of work.

M. N. SMITH-PETERSEN, M.D.

REPORT

In selecting the members of this Sub-Committee for the further study of internal fixation in the treatment of fresh fractures of the neck of the femur, care was taken to appoint men who were thoroughly familiar with the two methods of internal fixation in most common use, namely, the Smith-Petersen nail and multiple wires or pins. Further, in order that any personal element might be eliminated in the conclusions drawn, no man was asked to serve who had originated a particular method of treatment.

A preliminary report of 1485 cases compiled from tabulated data submitted by members of the Academy was made by Dr. H. Earle Conwell, Secretary of the Fracture Committee, at the Academy Meeting in January 1939, and published in *The Journal of Bone and Joint Surgery* (XXI, 483, April 1939). Unfortunately, that report did not constitute a true analysis of the cases according to the rules established by the present Committee, since it was not based upon an actual study of the roentgenograms and other data submitted.

For this report, the Committee has accepted only cases in which clinical and roentgenographic data were available. Opinion regarding the final results was based solely upon roentgenographic evidence; no written statements as to the status of union were considered. In addition to clinical data submitted on the tabulated forms provided by the Academy, roentgenograms were available in 923 cases. Although in a large proportion of these the roentgenograms were inadequate for study of the final results, many

* Presented at the Annual Meeting of the American Academy of Orthopaedic Surgeons, New Orleans, Louisiana, January 14, 1941.

interesting observations were made from the review of the entire group. The requirements for inclusion in the final analysis were as follows:

1. The fracture must be a complete, fresh fracture of the neck of the femur with displacement of the fragments.

2. Conclusive evidence of such a fracture must be presented, preferably by roentgenograms made prior to reduction. Without preoperative roentgenograms, the Committee was at times unable to distinguish a complete fracture of the femoral neck from an impacted fracture or a trochanteric fracture.

3. Roentgenograms must be submitted of the fracture after reduction, showing the type and position of the internal fixation employed.

4. Roentgenograms must be submitted of the fracture region one year or longer following reduction. Cases wherein final roentgenograms were made before the elapse of one year were eliminated, regardless of the status of union. A number of fractures which appeared to have united solidly in less than one year, and even after one year, have subsequently terminated in non-union. In order to eliminate error in so far as possible, one year was decided upon as a minimum follow-up period.

Because of the lack of data submitted, the number of cases which fulfilled these strict requirements was comparatively small. It was believed, however, that a study of a small number of complete cases with definite end results would be more valuable than the study of a larger series based upon less accurate data.

TABLE I

TYPES OF FIXATION EMPLOYED IN 241 COMPLETE CASES

Type of Fixation	Cases	Per Cent.
1. Smith-Petersen nails	144	59.8
2. Wires	83	34.4
3. Miscellaneous	14	5.8
Total	241	100.0

The data presented in Tables I to IV are based on the study of 241 cases wherein the criteria outlined above were fulfilled. The data contained in Tables V to VIII are based upon a study of the 923 cases.

The Smith-Petersen nails included all modifications of the three-flanged nail which were submitted to the Committee. The wires included multiple pins or wires, such as Austin Moore pins, Knowles pins, Gaenslen spikes, Kirschner wires, etc.; the majority consisted of Austin Moore pins. It was felt that the series was too small to differentiate between the various types of pins and wires. The miscellaneous group included screws, bone grafts, and other materials.

Solid bony union took place in 169, or 70.1 per cent., of the 241 accepted cases.

As stated before, only fractures wherein union was solid, as demonstrated by roentgenograms taken one year or longer after fixation was inserted, were accepted. If, in the

TABLE II

PERCENTAGE OF BONY UNION OBTAINED WITH EACH TYPE OF FIXATION

Type of Fixation	Cases	Unions	Per Cent.
1. Smith-Petersen nails	144	105	72.9
2. Wires	83	52	62.7
3. Miscellaneous	14	12	85.7
Total	241	169	70.1

TABLE III

INCIDENCE OF ARTHRITIC CHANGES FOLLOWING BONY UNION

Grade of Change	Smith-Petersen Nail		Multiple Wires	
	Cases	Per Cent.	Cases	Per Cent.
No change	77	73.4	37	71.2
Grade I	3	2.9	5	9.6
Grade II	5	4.7	2	3.8
Grade III	5	4.7	1	1.9
Grade IV	15	14.3	7	13.5
Total	105	100.0	52	100.0

opinion of the Committee, union was doubtful, the fracture was regarded as ununited. It is probable that some of those thus excluded will ultimately firmly unite. The 70.1 per cent. of bony unions indicated in Table II stood the test of a hypercritical committee and the handicaps associated with the development of a new method. It should be borne in mind that the proper insertion of fixation material for fractures of the neck of the femur requires skill and experience. This series included many of the earliest cases in which internal fixation was used; without doubt, therefore, in the hands of experienced surgeons, a higher percentage of bony unions may be expected in patients being treated at the present time.

The arthritic changes observed in the head were graded as follows: Slight arthritic changes not present in the original roentgenograms, but apparent one year or longer following reduction were considered Grade I; moderately advanced arthritic changes without deformity of the head were classified as Grade II; arthritic changes associated with flattening and definite changes in the contour of the head were classified as Grade III; and extensive arthritic changes associated with disintegration of the head were considered Grade IV.

The more severe arthritic changes, Grades III and IV, were observed eighteen months to two years following internal fixation. Severe changes with disintegration of the head of the femur (Grade IV) were usually associated with aseptic necrosis of the femoral head, as indicated by an increase in density in the roentgenograms. In two cases there was a marked enlargement and irregularity of the femoral head and neck. This may have been the result of an unusual proliferative reaction of the bone to metal, or may have been some other type of pathological process.

TABLE IV

THE RELATION OF REDUCTION TO UNION

Reduction	Total		Union		Non-Union	
	Cases	Per Cent.	Cases	Per Cent.	Cases	Per Cent.
Good	173	71.8	132	76.3	41	23.7
Fair	44	18.2	26	59.1	18	40.9
Poor	24	10.0	11	45.8	13	54.2
Total	241	100.0	169	70.1	72	29.9

Anatomical replacement of the fragments or a slight valgus position was regarded as a good reduction; apposition of 75 per cent. or more of the fractured surface of the neck with the corresponding fractured surface of the head, with only slight or moderate rotation of the head, was considered a fair reduction; apposition of less than 75 per cent. of the two fractured surfaces, or marked rotation of the head, or appreciable coxa vara, or a combination of these conditions, was considered a poor reduction.

The foregoing statistics are based upon the 241 complete cases. An additional study was made of the 682 cases in which roentgenographic and clinical data were insufficient to permit their inclusion in the final analysis. The following facts were obtained from a review of the entire series of 923 cases.

In the total of 923 cases, there were 107 deaths, or a total mortality of 11.6 per cent. In ninety cases, reduction and nailing were carried out in conjunction with an arthrotomy. Among these, there were nine deaths, a mortality of 10.0 per cent. The fixation was inserted without opening the hip joint in 600 cases; in this group there were sixty-nine deaths, a mortality of 11.5 per cent. In 107 cases the method of fixation was not stated; among these there were twenty-three deaths, a mortality of 21.5 per cent. The above data appear to indicate that an arthrotomy or open reduction does not increase the mortality. This, however, is probably not true. The patients upon whom an arthrotomy was done were principally private patients, whereas a large proportion of those whose fracture was fixed without arthrotomy were reported from city hospitals, and some of them from insane asylums. The general medical condition of the latter types of patients is poorer and the postoperative care inferior to that of private patients.

Of the 923 cases, 126 were rejected because the fractures were trochanteric in type, or the condition was a slipped upper femoral epiphysis rather than a fracture, or because of meager roentgenographic data. The following statistics were based upon the remaining 797 cases:

TABLE V

TYPE OF FIXATION EMPLOYED

Type of Fixation	Cases	Per Cent.
1. Smith-Petersen nails	572	71.8
2. Wires	162	20.3
3. Miscellaneous	63	7.9
Total	797	100.0

TABLE VI

FAILURE TO MAINTAIN REDUCTION

	Cases	Failures	Per Cent.
1. Smith-Petersen nails	572	63	11.0
2. Wires	162	34	21.0
3. Miscellaneous	63	13	20.6

A study was made to determine the efficiency of the fixative agent in maintaining the reduction of the fracture, whether or not the fixative agent broke, and the incidence of migration of the fixative agent either toward the acetabulum (intrusion), or toward the greater trochanter (extrusion).

Of the two cases in which the Smith-Petersen nail broke, reduction was maintained in one and lost in the other. One or more wires were broken in twenty-eight of the 162 cases in which they were used. Reduction was lost in twelve of these and was held in

TABLE VII

INCIDENCE OF BREAKAGE OF FIXATIVE AGENT

	Cases	Broken	Per Cent.
1. Smith-Petersen nails	572	2	0.35
2. Wires	162	28	17.3
3. Miscellaneous	63	2	3.2

TABLE VIII

MIGRATION OF THE FIXATIVE AGENT

	Cases	Migration	Per Cent.
1. Smith-Petersen nails	572	75	13.1
(a) Intrusion—29 (38.7 per cent.)			
(b) Extrusion—46 (61.3 per cent.)			
2. Wires	162	45	27.8
(a) Intrusion—29 (64.4 per cent.)			
(b) Extrusion—16 (35.6 per cent.)			

the remaining sixteen. Reduction was lost in both cases of the miscellaneous group in which the fixative agent broke.

Intrusion of either the Smith-Petersen nail or a wire to the point of contact with the acetabulum will result in erosion of cartilage and bone, thus leading to arthritic changes in the acetabulum. Marked erosion of bone was observed about wires which had migrated into the acetabulum. A single migrating wire tends to penetrate deeper into the acetabular floor than the larger Smith-Petersen nail. Extrusion of the Smith-Petersen nail sufficiently to lose its hold in the capital fragment will result in loss of reduction. Extrusion of the wires may or may not result in loss of fixation, depending upon the extent of extrusion and the number of wires involved. The tendency to migration of the wires was approximately twice that of the Smith-Petersen nails. The incidence of extrusion of the Smith-Petersen nails was approximately twice that of intrusion, whereas the incidence of intrusion of the wires was approximately twice that of extrusion.

It is the opinion of this Committee that, in the hands of experienced surgeons, the end results obtained in fractures of the neck of the femur have been greatly improved by the use of internal fixation. When employed by the inexperienced or untrained, the outcome is often deplorable and the end results inferior to those seen following non-operative methods.

Respectfully submitted,

WILLIS C. CAMPBELL, M.D., *Chairman*
H. EARLE CONWELL, M.D., *Secretary*
LAWSON THORNTON, M.D.
FRANK D. DICKSON, M.D.
HERMAN C. SCHUMM, M.D.

News Notes

The Fifty-Fifth Annual Meeting of **The American Orthopaedic Association** will be held in Toronto, Ontario, June 9, 10, 11, and 12, at the Hart House and the Royal York Hotel. Dr. D. E. Robertson, President, is in charge of the meeting, and careful plans have been made for the entertainment of the members and guests.

A clinical program will be presented on Monday and Tuesday morning by local surgeons.

At the session on Wednesday afternoon there will be a symposium on subjects related to the War. The scientific program is not yet completed, but copies will be mailed to members in the near future.

At the examination of the **American Board of Orthopaedic Surgery** held in New Orleans on January 11 and 12, 1941, forty-two candidates were examined and certificates were granted to twenty-eight.

The examination fees have been adjusted so as to be in closer accord with those of other specialty boards. A fee of $50.00 will accompany the application and cover the expenses of the first examination. Candidates who have to appear before the Board at second and third examinations will be charged additional fees of $25.00 for each examination following the first. This change took effect as of January 15, 1941, and is not retroactive.

The personnel of the Board at present is:

Dr. Fremont A. Chandler, Chicago, Illinois, *President.*
Dr. John C. Wilson, Los Angeles, California, *Vice-President.*
Dr. Guy A. Caldwell, New Orleans, Louisiana, *Secretary-Treasurer.*
Dr. George E. Bennett, Baltimore, Maryland.
Dr. Frank D. Dickson, Kansas City, Missouri.
Dr. Melvin S. Henderson, Rochester, Minnesota.
Dr. Samuel Kleinberg, New York, N. Y.
Dr. J. S. Speed, Memphis, Tennessee.
Dr. Philip D. Wilson, New York, N. Y.

The American Physiotherapy Association will hold its Twentieth Annual Convention at Asilomar, California, July 13 to 18, 1941.

Dr. Ralph M. Carter announces the association with him of Dr. S. S. Houkom. Their office is located at 607–610 Bellin Building, Green Bay, Wisconsin.

Dr. Donald C. Durman announces the association with him of Dr. Harold E. Mayne and the opening of a new office at 408 South Jefferson Avenue, Saginaw, Michigan.

The **American Academy of Physical Medicine** will hold its Nineteenth Annual Meeting and Scientific Session on April 28, 29, and 30, 1941, in New York, with headquarters at the Hotel Pennsylvania.

The Seventieth Annual Meeting of the **American Public Health Association** will be held at Atlantic City, New Jersey, October 14, 15, 16, and 17. Headquarters for the meeting will be the Convention Hall, and residence headquarters will be the Hotel Traymore.

The **International College of Surgeons,** of which Dr. Fred H. Albee is President, will hold its Fifth International Assembly in Mexico City, August 10 to 14, 1941, in response to the invitation of the Mexican Government. With the assembly will be scientific exhibits of the latest advances in surgery, and commercial demonstrations of the newest equipment. Information about the meeting may be obtained from Dr. Max Thorek, Executive Secretary, 850 West Irving Park Boulevard, Chicago.

At the Annual Founder's Day Dinner of the Kappa Sigma fraternity, in Washington, D. C., on December 10, Dr. Fred H. Albee, President of the International College of Surgeons, was presented with a bronze plaque. The presentation was made by Senator Warren R. Austin of Vermont. The award was made in recognition of Dr. Albee's services as Chairman of the New Jersey Rehabilitation Commission, which he helped establish, and for his outstanding achievement in orthopaedic surgery.

The First Annual Medical Meeting of **The National Foundation for Infantile Paralysis** was held at the Waldorf-Astoria Hotel, New York, N. Y., on November 7 and 8, 1940. Attending the meeting were the members of the medical advisory committees, the grantees of the Foundation, and the Board of Trustees. Reports of the activities of committees and grantees for the preceding year were presented, and recommendations were made for grants for 1940–1941.

The Foundation is concerned with promotion and furtherance of research on all phases of infantile paralysis. Studies are being carried on through grants from the Foundation on problems of epidemiology, virus research, relationship of nutrition to poliomyelitis, and the prevention and treatment of the disease. In addition, a program of professional and lay education has been promoted.

The following chairmen of medical committees were appointed:

Committee on Virus Research: Dr. Thomas M. Rivers, New York, N. Y.

Committee on Research for the Prevention and Treatment of After-Effects: Dr. Philip Lewin, Chicago, Illinois.

Committee on Nutritional Research: Dr. James S. McLester, Birmingham, Alabama.

Committee on Epidemics and Public Health: Dr. Herman N. Bundesen, Chicago, Illinois.

Committee on Education: Dr. Max M. Peet, Ann Arbor, Michigan.

Committee on Medical Publications: Dr. Morris Fishbein, Chicago, Illinois.

The Annual Meeting of the **British Orthopaedic Association,** originally scheduled for October 25 and 26, was held in Oxford on January 3, under the Presidency of Prof. T. P. McMurray.

The program included the following papers:

Late Results of Fractured Necks of Femur—Mr. A. L. Eyre-Brooke and Mr. K. H. Pridie.

Nerve Regeneration and Nerve Grafting—Mr. J. L. Young.

The Indications for Excision of the Patella—Mr. Norman Roberts.

The Correction of Scoliosis—Mr. G. E. Thomas.

Treatment of Fractures of the External Tuberosity of the Tibia—Mr. W. S. Diggle.

The Result of Radical Treatment in Diseases and Injuries of the Carpal Semilunar—Mr. F. C. Dwyer.

Treatment of Compound Fractures of the Leg—Mr. G. K. McKee.

The Organization of Orthopaedics in the R.A.F.—Mr. R. Watson-Jones.

Prof. H. J. Seddon was elected Honorary Secretary, succeeding Mr. E. P. Brockman.

The following were elected to Associate Membership:

Mr. James Rowan Armstrong.

Mr. Andrew L. Butler.

Mr. Ian Lawson Dick.
Mr. Erwin Flatow.
Mr. Edward John Gallagher.
Mr. Ronald Neville Houlding.
Mr. G. A. Nicholl.
Mr. Archibald Ronald.
Mr. Nicholas Vere-Hodge.
Dr. G. F. Pennell (Toronto, Ontario, Canada).

The following Honorary Member was elected:

Dr. José Trueta.

THE AMERICAN ACADEMY OF ORTHOPAEDIC SURGEONS

The Ninth Annual Convention of The American Academy of Orthopaedic Surgeons was held at the Hotel Roosevelt, New Orleans, January 12, 13, 14, 15, and 16, 1941, under the Presidency of Dr. Robert D. Schrock. The attendance was larger than at any previous meeting.

On the morning of January 13, a clinical program was presented at Tulane Medical School, with an address of welcome by Dean Maxwell E. Lapham. The afternoon of this day was free for the delegates to visit the scientific exhibits, which throughout the meeting were an outstanding feature.

The scientific program was presented at the sessions on Tuesday, Wednesday, and Thursday, and included the following papers:

Tuesday, January 14

Morning Session

Localized Fibrocystic Disease of Bone—Dr. Rufus H. Alldredge, New Orleans, Louisiana.

Aseptic Necrosis and Bone Drilling—Dr. Ernst Bergmann and Dr. Arthur H. Krida, New York, N. Y.

Operative Treatment of Legg-Perthes Disease—Dr. Paul B. Steele, Pittsburgh, Pennsylvania.

Thrombophlebitis and Postphlebitic Oedema—Dr. Alton Ochsner, New Orleans, Louisiana.

Congenital Pseudarthrosis—Dr. Harold B. Boyd, Memphis, Tennessee.

Shelf Operation at the Hip: New Method—Dr. Edwin W. Ryerson, Chicago, Illinois.

An Evaluation of Physiotherapy in Early Treatment of Anterior Poliomyelitis—Dr. H. R. McCarroll and Dr. C. H. Crego, Jr., St. Louis, Missouri.

Afternoon Session

Bone Tumors in Reference to Their Radio Sensitivity—Dr. Murray M. Copeland, Baltimore, Maryland.

Gradation of Ewing's Tumors—Dr. Willis C. Campbell and Dr. J. F. Hamilton, Memphis, Tennessee.

Hemangioma of the Vertebra—Dr. R. K. Ghormley and Dr. A. W. Adson, Rochester, Minnesota.

End Results of 200 Cases of Suppurative Arthritis—Dr. John A. Heberling, Pittsburgh, Pennsylvania.

The Use of Neo-Arsphenamine in the Treatment of Acute Osteomyelitis with Associated Staphylococcus Septicaemia: A Clinical and Experimental Study—Dr. Edward LeCocq, Seattle, Washington.

Wednesday, January 15

Morning Session

An Accurate Method for the Estimation of the Expected Growth of the Femur and Tibia in Individual Children—Dr. Gerald G. Gill, San Francisco, California.

A Study of End Results in Bone Lengthening—Dr. Beveridge H. Moore, Chicago, Illinois.

The Timing of Fracture Healing Process: Its Influence on Choice and Application of Treatment Methods—Dr. Clay Ray Murray, New York, N. Y.

Treatment of Osteomyelitis with Sulfathiazole Used Systemically and Locally—Dr. Frank D. Dickson, Dr. Rex L. Diveley, and Dr. Richard Kiene, Kansas City, Missouri.

The Fate of Fascia Lata in Knee-Joint Reconstruction (Histological Study)—Dr. E. T. Evans, Minneapolis, Minnesota.

Comparison of Stainless Steel and Vitallium as Material for Internal Fixation of Bone—Dr. J. Albert Key, St. Louis, Missouri.

Use of Vitallium Ferrule to Alleviate Lesions of the Head of the Radius—Dr. Kellogg Speed, Chicago, Illinois.

Fracture of the Patella Treated by Removal of the Fragments: End Results—Dr. J. E. M. Thomson, Lincoln, Nebraska.

Some Mechanical Derangements of the Knee—Dr. Allen F. Voshell and Dr. Otto C. Brantigan, Baltimore, Maryland.

Afternoon Session

Organization for Evacuation and Treatment of War Casualties—Col. N. T. Kirk, Washington, D. C.

President's Address—Dr. Robert D. Schrock, Omaha, Nebraska.

Conservative and Operative Treatment of Fractures of the Carpal Scaphoid—Dr. Keene O. Haldeman and Dr. Ralph Soto-Hall, San Francisco, California.

Use of Preserved Bone Grafts in Orthopaedic Surgery—Dr. Alberto Inclán, Havana, Cuba.

Treatment of Calcified Tendinitis—Dr. Joseph E. Milgram, Brooklyn, New York.

The Public Care of the Physically Handicapped—Dr. Fred H. Albee, New York, N. Y.

THURSDAY, JANUARY 16

Morning Session

The Rib Joints—Dr. Joel E. Goldthwait, Boston, Massachusetts.

The Rôle of Curare in the Prevention of Fractures in Convulsive Metrazol and Insulin-Shock Therapy—Dr. W. R. Hamsa, Omaha, Nebraska.

Experimental Studies in the Use of a U Clamp for Approximation and Fixation of Spinous Processes in Vertebral Fractures—Dr. Robert V. Funsten, Charlottesville, Virginia.

Surgery of the Intrinsic Muscles of the Hand Other Than Those of Opposition of the Thumb—Dr. Sterling Bunnell, San Francisco, California.

A New Operation for Acromio-Clavicular Dislocation—Dr. E. B. Mumford, Indianapolis, Indiana.

On Wednesday evening, January 15, the annual banquet was held at the Hotel Roosevelt. After the banquet, prizes were awarded by the Committee on Scientific Investigation for the Scientific Exhibits, as follows:

Gold medal to Dr. Clay Ray Murray and Dr. Stephen Hudack, for Originality of Presentation and Research. Their exhibit was entitled "Operative Fixation of Long-Bone Fractures".

Gold medal to Dr. Carl E. Badgley and Dr. George Hammond, for Scientific Information, as shown by their exhibit on "Suppurative Hip-Joint Disease".

Gold medal to Dr. Allen F. Voshell and Dr. Otto C. Brantigan, for the Clinical Value of their exhibit, "Internal Derangements of the Knee—Anatomical Demonstrations".

Certificates of Honorable Mention were awarded to the following for their exhibits:

Dr. Vernon L. Hart—Congenital Hip Without Dislocation.

Dr. Paul B. Steele—New Operative Treatment for Perthes' Disease.

Dr. Paul C. Colonna and Dr. Denman C. Hucherson—A Survey of Paralytic Scoliosis in Oklahoma.

Dr. Lenox D. Baker and Dr. Alfred R. Shands—Gross Pathology of Bone.

Dr. Guy A. Caldwell and Dr. Rufus H. Alldredge—Tibiofibular Diastasis and Associated Lesions.

Dr. Willis C. Campbell and Dr. Harold B. Boyd—Treatment of Fracture of the Ulna with Dislocation of the Head of the Radius.

Executive Sessions were held on Tuesday at noon and on Thursday morning. The new officers and the following members of committees were elected:

Membership Committee: Dr. Mather Cleveland, New York, N. Y.

Program Committee: Dr. James E. M. Thomson, Lincoln, Nebraska.

Committee on Scientific Investigation: Dr. G. E. Haggart, Boston, Massachusetts.

Committee on Legislation and Medical Economics: Dr. A. Bruce Gill, Philadelphia, Pennsylvania.

Representatives chosen for the American Board of Orthopaedic Surgery:

Dr. Joseph A. Freiberg, Cincinnati, Ohio, and Dr. Edward L. Compere, Chicago, Illinois.

Dr. Henry W. Meyerding, Rochester, Minnesota, was elected Representative of the Academy on the Board of Governors of the American College of Surgeons.

The Membership Committee announced the election of the following new members:

Dr. Lenox D. Baker, Durham, North Carolina.
Dr. Charles H. Baldwin, Utica, New York.
Dr. William H. Barker, Houston, Texas.
Dr. Harold B. Boyd, Memphis, Tennessee.
Dr. Louis W. Breck, El Paso, Texas.
Dr. Norman R. Brown, Spokane, Washington.
Dr. Wilfred S. Clark, Ventura, California.
Dr. John R. Cobb, New York, N. Y.
Dr. Harold H. Cohen, New York, N. Y.
Dr. James Pierce Cole, Buffalo, New York.
Dr. Malcolm B. Coutts, New York, N. Y.
Dr. Henry B. Crawford, Rochester, New York.
Dr. Edward H. Crosby, Hartford, Connecticut.
Dr. Clyde W. Dawson, Columbus, Ohio.
Dr. Otto A. Engh, Washington, D. C.
Dr. Louis A. Goldstein, Rochester, New York.
Dr. William T. Green, Boston, Massachusetts.
Dr. Benjamin B. Greenberg, New York, N. Y.
Dr. Edward J. Haboush, New York, N. Y.
Dr. Pinckney Harral, San Francisco, California.
Dr. Herbert W. Harris, Ann Arbor, Michigan.
Dr. Thomas Horwitz, Philadelphia, Pennsylvania.
Dr. Gilbert T. Hyatt, Fall River, Massachusetts.
Dr. Charles Edwin Irwin, Warm Springs, Georgia.
Dr. Frank S. Jones, Hartford, Connecticut.
Dr. Walter W. King, Peoria, Illinois.
Dr. Julius Kreitman, Brooklyn, New York.
Dr. Robert F. Legge, Oakland, California.
Dr. Max A. Levine, Los Angeles, California.
Dr. Herman S. Lieberman, New York, N. Y.
Dr. Frederick L. Liebolt, New York, N. Y.
Dr. Paul H. Martin, Jacksonville, Florida.
Dr. Robert Mazet, Jr., Great Neck, New York.

Dr. James McAteer, New York, N. Y.
Dr. H. Relton McCarroll, St. Louis, Missouri.
Dr. William D. McElroy, Youngstown, Ohio.
Dr. Donald S. Miller, Chicago, Illinois.
Dr. Ernest E. Myers, New York, N. Y.
Dr. Benjamin E. Obletz, Buffalo, New York.
Dr. Carson R. Reed, Jr., Shreveport, Louisiana.
Dr. Ward N. Rolland, Los Angeles, California.
Dr. Albert J. Schein, New York, N. Y.
Dr. Charles E. Sevier, Denver, Colorado.
Dr. John A. Siegling, Urbana, Illinois.
Dr. Frank A. Slowick, Pittsfield, Massachusetts.
Dr. Edward T. Smith, Houston, Texas.
Dr. Harry A. Smith, Wilkes-Barre, Pennsylvania.
Dr. Milton Strong Thompson, Portland, Maine.
Dr. Pedro Sanchez Toledo, Havana, Cuba.
Dr. James T. Tucker, Richmond, Virginia.
Dr. J. Irving Tuell, Seattle, Washington.
Dr. Raymond M. Wallerius, Sacramento, California.

The officers for the ensuing year are:

President: Dr. Oscar L. Miller, Charlotte, North Carolina.
President-Elect: Dr. Carl E. Badgley, Ann Arbor, Michigan.
Vice-President: Dr. Herman D. Schumm, Milwaukee, Wisconsin.
Treasurer: Dr. E. Bishop Mumford, Indianapolis, Indiana.
Secretary: Dr. Rex L. Diveley, Kansas City, Missouri.
Librarian-Historian: Dr. Philip Lewin, Chicago, Illinois.

The next meeting will be held in Washington, D. C., January 11 to 15, 1942. Dr. Guy W. Leadbetter, of Washington, was appointed chairman of the Local Arrangements Committee.

Current Literature

DIE NORMALE UND GESTÖRTE KNOCHENBRUCHHEILUNG (Normal and Pathological Fracture Healing). Dr. Werner Block. Stuttgart, Ferdinand Enke, 1940. 49 marks.

The present volume constitutes the sixty-second in the series of *Neue Deutsche Chirurgie* originally begun by von Bruns and continued under the editorship of Sauerbruch. For those interested in the healing of fractures, this book is of the utmost importance. As its title may be translated, it is not only a study of normal and pathological fracture healing, but also a study of the healing of fractures in normal and pathological bones. Indeed, more than this, it is a review, on a broad biological basis, of all the processes which lead to the development of bone.

The first section of the work is devoted to a rapid historical review of the various theories of fracture healing. Since this involves an evaluation of the importance of the different components of bone healing, the next eighty pages are dedicated to a consideration of the rôle of the periosteum, the marrow, the bone lamellae, the connective tissue, the muscles, the reticulo-endothelial system, the blood vessels, and hemorrhage, as well as the nerve tissue, in the mechanism of fracture healing. All of the evidence, both for and against any particular point of view, is presented with a singularly impartial and critical attitude, even though the personal preferences of the author are presented in short summaries.

In logical sequence, the peculiarities of healing in special types of fractures—for example, epiphyseal fractures, joint fractures, and fractures of the long and flat bones—are considered. The influence on healing of treatment by means of plaster, of traction, and of open operation is discussed. Despite the fact that the author is clearly in favor of the method of balanced traction, the opinions of proponents of other methods are clearly presented.

The chapters devoted to the explanation of the physics, the physical chemistry, the colloid chemistry, and the chemistry of fracture healing are among the most interesting and stimulating in the entire volume. Though the significance of the mechanical and physico-chemical influences are clearly delineated, the author frankly admits that some additional force, a "nisus formativus"—the old "vitalismus"—must be invoked to explain the fundamental problems, for which not even the newer knowledge of diet, vitamins, and hormones suffices.

In general, the normal appearance of bone is considered as a manifestation of normal differentiation in young mesenchymal tissues. The pathological manifestations are to be considered merely as an interference with the normal course of differentiation. It is in this light that the whole interesting chapter on pseudo-arthroses and the final chapter on fracture healing in diseased bone are approached.

All in all, this book is one of the most fascinating that has fallen to the lot of this reviewer. Not only is it a repository of much important and scattered data; it is a rich storehouse of bibliographical references and a perfect mine in which unending problems for further research lie exposed to the mind of those who are interested. The book would well repay translation for those who cannot read German, and is a "must" for those who can master the language difficulty.

DIAGNOSIS AND TREATMENT OF ARTHRITIS AND ALLIED DISORDERS. H. M. Margolis, M.D., F.A.C.P. New York, Paul B. Hoeber, Inc., 1940. $7.50.

General practitioners and medical students will obtain from this book a much better understanding of the many problems of "rheumatism" and arthritis as well as valuable information concerning the diagnosis and treatment of individual patients. The author strives particularly to dispel the attitude of defeatism which prevails among many general

practitioners responsible for the treatment of patients with arthritis. Poor therapeutic results, upon which the existence of this attitude depends, are due in part to errors in diagnosis and failure to prescribe appropriate therapy.

The classification of the arthritides is simple and workable. Nearly half of the book is devoted to atrophic or rheumatoid arthritis. Many readers will wish that the sections on hypertrophic arthritis, rheumatic fever, and the specific infectious arthritides had been presented in more detail. Even less space is allotted to traumatic arthritis, hemophilic arthritis, the arthritis of serum sickness, neuropathic joint disease, fibrositis, Dupuytren's contractures, and miscellaneous rheumatic conditions. Such brevity is unfortunate in that information often required by the practitioner has been omitted. A much larger section is devoted to pain in the shoulder and arm, subacromial bursitis, periarthritis of the shoulder, cervical rib, the scalenus anterior syndrome, and various types of nerve involvement. The last hundred-odd pages contain a comprehensive discussion of low-back and sciatic pain. The sections on pain in the shoulder, arm, and low back are of value principally from the point of view of differential diagnosis. The importance of considering "sciatica" as a symptom, rather than a diagnosis, is well brought out. Each chapter concludes with a bibliography, which in general is well selected.

The chapters dealing with the constitutional background and etiology of rheumatoid arthritis are highly speculative. The author's opinions are traditional, though he is generally careful to qualify statements for which direct proof cannot be given. It is admitted, for instance, that the conclusion that focal infections play a definite rôle in the etiology of rheumatoid arthritis is based solely on clinical impressions.

The clinical and roentgenographic descriptions of rheumatoid arthritis are detailed. There is no mention of eye lesions which represent a rare, yet sometimes the first, manifestation of this constitutional disease. There is a brief discussion of the sedimentation rate in which it is stated that corrections for anaemia are unnecessary. The author fails to refer to literature which upholds the opposite point of view. The importance of this point lies in the fact that certain patients with anaemia will be considered to have elevated sedimentation rates unless the corrections are taken into account.

For the general practitioner, the chapters on the treatment of rheumatoid arthritis should prove to be the most valuable in the book. Those dealing with the prevention and correction of deformities contain information seldom taught in medical schools or on the medical wards of hospitals. These chapters are well illustrated and the author's recommendations are logically presented. In the chapter on physical therapy, stress is correctly placed upon simple and effective methods of treatment which the patient or his family can carry out at home. Although the author describes in detail the technique of roentgen-ray therapy, he admits that its value remains to be proved. Concerning the value of gold as an adjunct in the treatment of rheumatoid arthritis, the author is hopeful, but not completely convinced. Since clinical data relative to the effectiveness of gold are still incomplete, this opinion is shared by many others in this country. The use of colloidal sulphur, vaccines, fever therapy, foreign-protein therapy, or bee venom in the treatment of rheumatoid arthritis is not recommended.

DIE ANGEBORENE HÜFTVERRENKUNG ALS ORTHOPÄDISCH-GEBURTSHILFLICHES PROBLEM (Congenital Dislocation of the Hip as an Orthopaedic-Obstetrical Problem). Hans Storck. (Beilageheft zur *Zeitschrift für Orthopädie*, LXX.) Stuttgart, Ferdinand Enke, 1940. 11 marks.

A study of the etiology of congenital dislocation of the hip must include a consideration of the frequent association of scoliosis, breech presentation, torticollis, and dislocation of the knee. These all point to the presence of a mechanical factor which is the restricted space in the uterus. The usual manifestation is the abnormal position of the foetus,—breech presentation in half of the cases of wry-neck, and in one-fifth of all cases of congenital dislocation of the hip. Obstetrical abnormalities must be considered as pri-

mary, and orthopaedic deformities as secondary. Breech presentations are commonly associated with cranial asymmetry, less frequently with true torticollis, and with some disturbance of the formation of the hip joint. The associated orthopaedic deformities must be due in some cases to abnormalities of intra-uterine position.

The hereditary nature of these deformities is explained by hereditary mechanical abnormalities of intra-uterine position. This probability is based upon the frequent association of hip dislocation and torticollis with breech presentation, as proved by statistics.

CLINICAL PELLAGRA. Seale Harris, M.D., and Seale Harris, Jr., M.D. St. Louis, C. V. Mosby Co., 1941. $7.00.

This monograph is based on the personal views and experiences of a southern physician who has observed pellagra since the days when this disease was first generally recognized in the southern states. As an historical account, it is of interest because it deals at length with the many earlier beliefs and hypotheses concerning the etiology of pellagra. Some of these hypotheses, the author feels, are still worthy of serious consideration, since, in his view, the more recent theory of dietary deficiency does not provide a complete explanation of the causation of this disease. In describing the pathology of pellagra, the author presents certain views which are frankly based on individual opinion and cannot be said to have achieved general acceptance at the present time. This is particularly true of his views concerning the rôle of intestinal toxins and liver insufficiency in the production of the endemic disease. His clinical description of pellagra, which is accompanied by some good illustrations, should be found useful, particularly by those who wish to obtain some knowledge of the less common manifestations of the disease.

The final chapters are devoted to the discussion of the problem of pellagra prevention in the South. Almost every one working on nutritional problems will agree with the authors' emphasis on the importance of a good general diet in this connection, but some may not agree with their disapproval of providing the vitamin-B complex in the form of yeast, a therapeutic agent which they stigmatize as "this fungus".

The book contains sections by other authors, including two chapters summarizing the investigative work that has been carried out in recent years at Duke University and the University of Georgia. These chapters, written by Drs. Smith and Ruffin, and by Dr. Sydenstricker, are particularly interesting and informative.

The extensive bibliography renders the book particularly useful for reference purposes.

TREATMENT IN GENERAL PRACTICE: SURGERY (*Continued*). Articles republished from the *British Medical Journal*, Vol. IV. London, H. K. Lewis & Co., Ltd., 1940. 16 shillings net.

Between 1936 and 1938, three volumes were published under the title, "Treatment in General Practice". The first two dealt with some of the major medical disorders, the third with anaesthesia and surgery in general practice. This fourth volume contains articles which have appeared in the *British Medical Journal* since 1938. This volume, with its three predecessors, gives a complete conspectus of surgery for the general practitioner, which will prove of value to him in the pursuance of his every-day work. This edition, if used alone, will serve a useful purpose, as it covers a wide range of surgical conditions.

The book appears at a time when the British surgeon's skill is being put to the acid test, as a result of the War, but surgical principles are essentially the same in war as in peace.

The volume contains 552 pages of text with 143 illustrations. The first eleven chapters deal with injuries and fractures of the shoulder joint, the upper arm, elbow, lower arm, and hand. Every major injury of importance, which might occur in the upper ex-

tremity, is covered in a systematic manner. The anatomy of the region is described, the mechanism of the fracture disclosed, and its immediate and after-care outlined. A chapter on the sulphonamide chemotherapy in surgical infections follows. Chapters on lesions in the breast, injuries to the chest and spine pursue the same general method of presentation. The treatment of joint injuries, with detailed descriptions of manipulations of difficult joints, is next taken up. Six chapters cover the field of surgery of the genito-urinary tract; five take up the lesions of the rectum and anus. Ten chapters deal with the lower extremity in a manner similar to that employed with the arm. In addition to descriptions of the fractures of the extremity, the subject of amputated stumps and artificial limbs is taken up, and the preferable amputation is emphasized. An excellent chapter deals with penetrating wounds of the joints.

These and many other subjects are covered in a methodical and clear manner. The book represents the opinions of a number of British surgeons who are authorities upon the field each has presented. In the main, the surgical principles laid down are the same the world over. The general practitioner will find this volume a valuable reference book. Here he can find quickly a clear and concise discussion of many of the surgical problems with which he is often faced.

ON ALBERS-SCHÖNBERG'S DISEASE (MARBLE BONES). S. van Creveld and N. I. Heybroek. *Acta Paediatrica* XXVII, 462, 1940.

Two cases of Albers-Schönberg disease (marble bones) are reported in detail by van Creveld and Heybroek of Amsterdam. The first case was that of a new-born female child. Pregnancy had been uneventful and birth had been normal. Enlargement of the heart to percussion led to roentgenographic examination and discovery of the disease. There was sclerosis of the entire skeleton with narrowing of the marrow spaces. The blood picture showed erythroblastemia, thrombopenia, increase in stab cells, juvenile forms, and myelocytes. The serum calcium was normal; the phosphorus and phosphatase were decreased. The child died on the ninth day. Autopsy showed a purulent meningitis, slight enlargement of the heart and spleen, and extramedullary blood formation. The bones showed a diffuse osteosclerosis with failure of resorption of cartilage in the endochondral ossification and retardation of resorption of the periosteal bone on the inner side. There were no normal blood-forming elements. Practically all of the marrow was replaced by large cells resembling reticulum cells.

The second patient, a girl of four and one-half years, was brought for roentgenographic examination because she had been blind since birth. Extensive chemical studies showed normal values for calcium, phosphorus, magnesium, and phosphatase. Fracture of the left femur occurred while the patient was under observation.

From a review of 127 cases reported, including their own, the authors conclude that the disease is congenital, but not hereditary; that bodily development is retarded, but mental development, as in their second case, is normal. There is an increased tendency to fractures, and a tendency to clubbing of the long bones; transverse bands parallel to the line of the epiphyses are common. There is no definite evidence of disturbance in any organ. Diagnosis can be made only by roentgenographic examination. The authors can suggest no possible etiology nor any therapy which favorably influences the disease.—*J. G. Kuhns, M.D., Boston, Massachusetts.*

ROENTGENOGRAPHIC SYMPTOMS OF MENISCAL LESION IN THE KNEE JOINT. Knut Lindblom. *Acta Radiologica*, XXI, 274, 1940.

The author studied a series of seventy-four cases in which arthrography was undertaken. The arthrogram was made following an injection of nine cubic centimeters of a 35-per-cent. solution of perabrodil. The author states that an arthrographic diagnosis of meniscal lesion was made in forty-three cases, operation subsequently carried out in twenty-seven, and the arthrographic diagnosis was verified in twenty-five. The symp-

toms of meniscal lesion shown by arthrogram consisted of contrast filling of a fissure in thirty-five cases; partial defect of the meniscus in ten cases; and interposition of the soft tissues between the condyles in two cases. A number of reproductions of roentgenograms are included to demonstrate the author's criteria for making the differential diagnosis from these three conditions. In twenty-seven of the cases in which arthrography was performed, a local osteophyte formation on the surface of the tibial condyle was noted. In twenty-four of these twenty-seven cases, a meniscal lesion was observed on the same side where the osteophyte was found. As a rule, osteophytes do not appear until months or years after injury to the cartilage has occurred; but in two cases, osteophytes were found as early as three weeks after the trauma. It is the author's opinion that the presence of the local osteophyte supports the clinical diagnosis of an early meniscal lesion. "In the majority of cases confirmation as to the presence of meniscal lesion can be obtained by arthrography. For the arthrographic diagnosis, the fissures of the meniscuses are most important."—*Henry Milch, M.D., New York, N. Y.*

THE GROSS PATHOLOGY OF SPONTANEOUS BONE TUMOURS IN MICE. F. C. Pybus and E. W. Miller. *The American Journal of Cancer*, XL, 47, 1940.

These authors have studied spontaneous bone tumors in a high-incidence strain of inbred mice. Nearly 600 bone lesions were found in more than 300 mice. The femora, tibiae, and spines were the commonest sites. The types of growth were extremely varied representing all possible phases in the activity of the osteoblast, from the development of true hard bone to spindle-cell sarcoma with widely disseminated metastases.

Careful analysis is presented of the age and sex incidence, distribution of metastases, clinical symptoms and signs, and gross and roentgenographic appearance. The report is illustrated with photographs, roentgenograms, and several tables.—*Grantley W. Taylor, M.D., Boston, Massachusetts.*

THE HISTOLOGY OF SPONTANEOUS BONE TUMOURS IN MICE. F. C. Pybus and E. W. Miller. *The American Journal of Cancer*, XL, 54, 1940.

This report describes the histological appearances encountered in a study of the tumors described in the preceding abstract, and is illustrated with numerous photomicrographs. The main classes of tumors are: hard osteoma, cancellous osteoma, osteosarcoma, osteosarcoma with giant cells, and chondro-osteosarcoma. Tumors in which bone and osteoid tissue predominate are the most frequent.—*Grantley W. Taylor, M.D., Boston, Massachusetts.*

INTERNAL DERANGEMENTS OF THE KNEE JOINT. AN ANALYSIS OF ONE HUNDRED CASES WITH FOLLOW-UP STUDY. L. Kraeer Ferguson and Wesley D. Thompson. *Annals of Surgery*, CXII, 454, 1940.

This is a review of 100 patients with internal derangement of the knee joint, who were operated upon, and the follow-up results in ninety-five. The anatomy, mechanism of injury, treatment, pathology, and end results are discussed.

The mechanism of injury is similar in nearly all cases,—that is, internal torsion of the femur on the tibia with the knee partially flexed. Eighty-two cases were in males, and 52 per cent. occurred in competitive sports. Sixty-eight were operated upon before their thirtieth year.

Primary treatment was always conservative. The average time from the original injury to operation was two and a half years. In the authors' experience, any knee with true locking eventually has to be operated upon.

Sixty-five of the 100 cases showed frank injuries to the menisci themselves. All but two were injuries to the internal semilunar. Thirty-one per cent. of cases had bucket-

handle tears, and 18 per cent. had tears in the anterior portion of the cartilage. In ten cases the tears were in the middle portion, and in six, in the posterior portion, of the cartilage.

In thirteen of the patients operated upon, there was a definite looseness of the cartilage. Frequently associated with this was a definite hypertrophy of the infrapatellar fat pad. These cases presented no characteristic diagnostic symptoms. The authors recommend excision of both fat pad and cartilage. There were sixteen cases in which only hypertrophy of the fat pad was found. These patients had pain and effusion. In most cases there was a history of limitation of extension.

Results were good in four cases of injury to the anterior crucial ligaments, despite the fact that in only one was an attempt at repair made. This seems most extraordinary.

There were five cases in which osteochondritis dissecans or foreign bodies were found. The abstractor presumes that the authors use the term foreign bodies to designate cartilaginous or osteocartilaginous loose bodies. The results were good in this group.

The essential points in the plan for operation and after-care are: no special preparation of knee, spinal anaesthesia, use of a tourniquet, loose suture of synovial capsule, pressure bandage without splint, early ambulatory after-care with early weight-bearing.

The results were excellent in a very high percentage of cases.—*O. B. Bolibaugh, M.D., San Francisco, California.*

FACTORS INFLUENCING THE PROGNOSIS IN OSTEOGENIC SARCOMA. Bradley L. Coley and John L. Pool. *Annals of Surgery,* CXII, 1114, 1940.

This study is based on 217 cases of osteogenic sarcoma observed at the Memorial Hospital and attempts to evaluate the factors influencing the prognosis. Microscopic confirmation was obtained for 160 of this group. Thirty-five patients, or 22 per cent., of this latter group remain free of disease five years after treatment.

The factors considered were: age, sex, interval elapsing between onset of symptoms and amputation of limb, anatomical location of tumor, biopsy, histological grading of tumor, and nature of treatment employed.

The peak of the age incidence occurs in late adolescence. The prognosis is exceedingly bad in this group, and nearly as bad in patients over forty. When the lesion is associated with Paget's disease, the prognosis is also extremely bad.

Of the 160 cases 100 were males and sixty females. The survival rate in females was 28 per cent., compared with 18 per cent. in males. The authors were unable to account for this difference.

Pathological fracture occurred in fifteen cases. In fatal cases the duration of life was halved. Early pathological fracture was seen most often in rapidly growing malignant tumors.

There was an interval of one year or more between onset of symptoms and amputation in 18 per cent. of the patients who died and in 22 per cent. of those who survived. Delay was usually due to failure to make a correct diagnosis.

The lower third of the femur is the most frequent location of the tumor, with the upper end of the tibia, the humerus, and fibula next in order of frequency. The more peripheral the site of the tumor, the better the prognosis.

The authors recommend aspiration biopsy and state that it was successful in 75 per cent. of cases in which it was attempted.

The gross pathological picture, as well as the microscopic picture, determine the malignancy of the tumor. The survival rate for low-grade malignancies was 40 per cent.; for average, 16 per cent.; and for high-grade malignancies 15 per cent.

Of the patients in whom amputation was performed through the bone primarily involved, 35 per cent. survived five years. Only 21 per cent. survived amputation by disarticulation through the joint above, and 36 per cent. survived amputation through the bone above. The authors believe that indiscriminate disarticulation of the hip joint for tumors in the lower third of the femur is not justified.

Amputation above with preoperative radiation remains the method of choice. Radiation alone was used in thirty-two cases, with only one survival.

Coley's toxins were administered in ninety-one cases, with twenty survivals. The authors feel that they are definitely indicated during post-operative period in young individuals.—*O. B. Bolibaugh, M.D., San Francisco, California.*

SACRO-ILIAC TUBERCULOSIS. H. J. Seddon. *British J. Surg.*, XXVIII, 193, 1940.

A group of 176 cases of sacro-iliac tuberculosis have been reviewed. From this study it is shown that it is a disease of young adults. In 33 per cent. of the cases an isolated lesion without sinuses was found. In 31 per cent. the lesion was still isolated, but sinuses were present. Tuberculosis elsewhere in the body was found in 36 per cent. of the group.

Abscesses formed in 72 per cent., and the pain usually subsided with the formation of the abscess. Associated lesions occurred chiefly in the lungs, joints, and lumbar spine. Variations in the severity of the disease and associated lesions were noted in different parts of the country. The commonest roentgenographic findings were erosion of the joint surfaces.

The mortality rate over a six-year period was 10 per cent. for the closed isolated lesions, 25 per cent. for the isolated lesions with sinuses, and 55 per cent. when other foci were present.

Relapses after recovery were rare and the patient was usually able to return to duty. Sinuses occasionally caused chronic invalidism. "Bony ankylosis is probably the usual end result of conservative treatment, even in the absence of secondary infection."

Conservatism should be used in the treatment. The surgical results are not encouraging. Fifty-four operations were performed on forty-nine patients. Radical excision was practiced twelve times with six successes. The Verrall type of fusion was done twenty-one times with satisfactory results in fourteen. Smith-Petersen's operation was done on fourteen patients, and the result was considered satisfactory in all but two.

Operation did not reduce the period of hospital treatment which averaged eighteen months.—*Ernest M. Daland, M.D., Boston, Massachusetts.*

THE TREATMENT, COMPLICATIONS, AND LATE RESULTS OF ACUTE HÆMATOGENOUS OSTEOMYELITIS BASED ON A STUDY OF 500 CASES ADMITTED TO THE LONDON HOSPITAL DURING THE YEARS 1919–37. E. C. B. Butler. *British J. Surg.*, XXVIII, 261, 1940.

Two-thirds of the patients in this study were males, and the majority were under twenty years of age. The staphylococcus was the commonest organism isolated. Great emphasis is laid on quantitative blood cultures in determining the prognosis and the progress of the disease.

One-fourth of the patients had osteomyelitis of the femora, and one-third showed involvement of the tibiae. About one-half of the patients made a complete recovery, but the others had deformity, pain, discharge, or recurrent infection.

Treatment may be summarized as follows: blood culture, estimation of the antibodies, intramuscular administration of staphylococcal antitoxin, absolute rest of the affected bone, an abundant supply of fluids, and then operation which may consist of aspiration, incision of the periosteum, or incision and drilling of the bone.

After operation the bone is kept at rest in plaster. Repeated small transfusions may be used. Careful watch is kept for infection in other bones and for local arthritis.—*Ernest M. Daland, M.D., Boston, Massachusetts.*

OBSERVATIONS ON DENTAL SEPSIS AND ITS RELATION TO DISEASES. M. D. Ananthachari. *Journal of the Indian Medical Association*, IX, 529, 1940.

The author comments wisely on the relation of oral sepsis to arthritis, peptic ulcer,

iritis, and anaemia. Recognizing the causative importance of dental infections in a wide variety of metastatic conditions, he yet counsels conservatism in the removal of teeth.—*Robert M. Green, M.D., Boston, Massachusetts.*

TRACTION LESIONS OF THE EXTERNAL POPLITEAL NERVE. Harry Platt. *The Lancet*, II, 612, 1940.

Mr. Platt further reviews and illustrates with additional cases a lesion which he described in *The Journal of Bone and Joint Surgery* in 1928. Nine examples of traction lesions of the external popliteal nerve produced by adduction injuries to the knee are reported. Of the nerve lesions, four were examples of overstretching without loss of continuity, and five were complete ruptures treated by end-to-end suture. The nerve sutures, performed after a delay of three months and longer, gave disappointing results. In traction lesions of this type, the injured nerve should, therefore, be explored within the first few weeks of the accident. In exploring the graver types of complete rupture the distal part of the nerve trunk should be sought in the region of the tear in the lateral capsule of the knee joint.—*Lenox D. Baker, M.D., Durham, North Carolina.*

STANDARD PATTERN FOR HIP SPICA. W. Sayle Creer. *The Lancet*, I, 9, Jan. 4, 1941.

The author describes a simplified method of applying a "creamed-fabric" plaster hip spica. His method is a modification of Trueta's pattern. The basic idea of the suggested modification is to use three pieces for the hip spica as in the shoulder spica. Several suggestions are offered to facilitate applying such a cast. From the number of articles appearing in the English literature on the use of patterns in applying plaster casts, the method is evidently an efficient and rapid procedure, and one which should be studied by those in America who are to have charge of such work in case of wartime or civil emergencies. To this group the article is highly recommended as it is concise and well illustrated.—*Lenox D. Baker, M.D., Durham, North Carolina.*

ALBAN KÖHLER'S DISEASE. I. N. Odesskiy and T. G. Babkin. *Novy Khirurgicheskiy Arkhiv*, XLV, 305, 1940.

The authors examined 144 normal children from three to eight years of age to establish whether this condition exists in normal children. A roentgenographic study was made and correlated with clinical and literary data. The investigation led the authors to the conclusion that Köhler's disease of the navicular bone is only a variety of normal development of the foot and does not represent a pathological entity.—*Emanuel B. Kaplan, M.D., New York, N. Y.*

JUVENILE OSTEOCHONDROSIS. C. A. Stammel. *Radiology*, XXXV, 413, 1940.

The author gives an excellent review of the condition of osteochondritis or "osteochondrosis", as more recently suggested by Harbin, because it involves various centers of bone growth. The author points out the confusion which has come from designating the lesions in their various locations, by the names of the men who first described them, and the desirability of classifying them all as osteochondrosis. He gives good descriptions of the condition in its various locations, with its course, symptomatology, roentgenographic characteristics, and final outcome, and with excellent illustrations.—*Edward N. Reed, M.D., Santa Monica, California.*

ROENTGEN FINDINGS IN CAISSON DISEASE OF BONE, WITH CASE REPORTS. Richard A. Rendich and L. A. Harrington. *Radiology*, XXXV, 439, 1940.

The authors review the literature on caisson disease of bone, and describe four cases from their own service.

The primary lesion is an accumulation of nitrogen gas in the bone, with a resultant massive aseptic necrosis. Involvement of the articular cortex and cartilage leads to the slow development of arthritis deformans, with or without osteocartilaginous loose bodies. Medullary calcification and disturbance of the lamellar structure of the ends of the larger long bones are characteristic.—*Edward N. Reed, M.D., Santa Monica, California.*

VERTEBRA PLANA (CALVÉ) (OSTÉOCHONDRITE VERTÉBRALE INFANTILE). Bruno Valentin. *Revista Brasileira de Cirurgia*, June 1940.

The effects of vertebra plana (Calvé) have been known for about fifteen years. Previously, it had been considered a tuberculous affection of the spine, but it should be classified among the diseases due to growth disturbance, together with diseases of the head of the femur, the navicular bone, etc. The author states that there have been only about thirty cases reported in the literature, and these occurred in children from two to fourteen years of age. He then discusses the clinical picture and the roentgenographic diagnosis of the disease. Regeneration of the affected vertebrae is evident at about the time that puberty begins.

The author reports two cases. One of his patients, who showed the affection in several vertebrae, was improving but had not entirely recovered.

RECONSTITUIÇÃO ARTICULAR AUTOPLASTICA DO COTOVELLO VALOIÇANTE (Autoplastic Articular Reconstruction of the Flail Elbow). Achilles de Araujo. *Revista Brasileira de Orthopedia e Traumatologia*, II, 3, 1940.

The author describes a case of elbow reconstruction in which, through a posterior incision, the elbow was approached, and a new articulation created on the anterior surface of the lower end of the humerus with a posterior ledge. The author believes that subperiosteal resections of the elbow are more useful than other types of operations in cases of traumatic lesions of the elbow, whether suppurative or not. The importance of physiotherapy is indicated. In the author's series of eleven patients with flail elbow, four were treated and improved by physiotherapy alone; two were treated by open capsuloligamentous reconstructions; two were operated upon to obtain bony ankylosis; and one required an amputation.—*Emanuel B. Kaplan, M.D., New York, N. Y.*

AN ANALYSIS OF THE TREATMENT OF SIXTY-EIGHT CASES OF FRACTURES OF THE SHAFTS OF THE TIBIA AND FIBULA WITH ROGER ANDERSON SPLINT. Howard A. Swart. *Southern Med. J.*, XXXIII, 1081, Oct. 1940.

After describing the method of using the splint in the treatment of fractures of the shafts of the tibia and fibula, the author makes a statistical analysis of the age, sex, color, and occupation of the patients; types and locations of fractures; incidence of infections; details of treatment; and results. There was slow union in 10.3 per cent. and non-union in 5.8 per cent. Open reduction was used in 3 per cent. Of the known end results 90.7 per cent. were good and 9.3 per cent. bad (non-union); 10.3 per cent. were still under observation. The average time away from work was 7.43 months. The author discusses common errors and the advantages and disadvantages of the method of treatment.—*Fred G. Hodgson, Atlanta, Georgia.*

PRACTICAL PROBLEMS OF PHYSICS IN ORTHOPEDIC SURGERY. Earl D. McBride. *Southern Med. J.*, XXXIII, 1272, Dec. 1940.

The orthopaedic surgeon must be proficient in the science of mechanics; he must be a construction engineer as well as a surgeon. The analytical study of physical disability involves more than clinical judgment and decision. The laws of physics, and mechanical demands must at least be recognized as dominating the biological laws. The forces of stress and equilibrium are accurately calculated for the construction of inanimate material. The same approach may be made to living material, even though it may not

reach perfection because of biological variations. Several examples are briefly presented here to illustrate how such information may be formulated mathematically.—*Fred G. Hodgson, M.D., Atlanta, Georgia.*

THE SURGICAL TREATMENT OF UNEQUAL LEG LENGTH. Paul H. Harmon and William M. Krigsten. *Surgery, Gynecology and Obstetrics*, LXXI, 482, 1940.

The writers discuss three methods of leg-length equalization, along with the prevention of shortening, and physiology of bone bearing on longitudinal growth disturbance. Most length discrepancies of one to five inches can be corrected by surgery.

The major causes of shortening are perforation of infection through epiphyseal lines and into joints, too long immobilization for poliomyelitis, and involvement of the epiphyses about the knee. Correct treatment of infection, poliomyelitis, and trauma will do much to reduce the incidence of inequality. Fusion of the upper femoral epiphysis causes little inequality if the patient is not immobilized too long. Unilateral lumbar ramisectomy has no consistent effect on longitudinal growth.

The three methods are: (1) operative slowing of growth, (2) shortening of the long extremity, and (3) lengthening of the short extremity. Operative slowing, the most conservative method, is done by epiphyseal arrest and has a definite field of usefulness, but is limited to the ages seven to twelve (fifteen in boys). Great care must be taken in the selection of cases and in considering the appropriate age for arrest. Operations should be done as early as possible. Growth factors need more study before real accuracy can be obtained. The operative technique of Phemister is described. One hundred and twenty patients have been operated upon with no infection or deaths.

Shortening of the long extremity is the most exact and applicable method. Several techniques are described, and that of making medullary and onlay grafts from the removed section of bone is recommended. Twenty-five of these operations have been done with excellent results in twenty-four. In one, infection developed, but later union was attained.

Lengthening of the short extremity has been done eleven times,—four times in the leg and seven times in the thigh; the results in three were unsuccessful. This is the least favored of the three methods because of technical difficulty and limitation in indications. With adequate musculature, and suitable bone (no previous osteomyelitis), equal extremity length should be obtainable. Multiple-pin methods with Z osteotomy for the leg and Compere's addition of a large tibial graft at the osteotomy site in the femur are recommended. Methods and results are illustrated by line drawings, photographs, and roentgenograms.—*Richard McGovney, M.D., Santa Barbara, California.*

FRACTURES IN THE NECK OF THE FEMUR—ACCURATE SUBCUTANEOUS FIXATION WITH SCREWS. Edwin O. Geckeler and Alfred Tuttle. *Surgery, Gynecology and Obstetrics.* LXXII, 106, Jan. 1941.

The writers describe a two-screw method of internal fixation which they have used in fifty cases since 1936. No cases of non-union occurred except in some with previously ununited fractures.

The method consists of subcutaneous insertion of two screws under fluoroscopic control through a guide and cannula apparatus. The guide prevents the screw from missing the abnormally anteverted neck. The apparatus is clearly described and illustrated. The anaesthetic is pentothal sodium. The reduction is by the Leadbetter method. The extremity is kept in wide abduction and internal rotation. The operator's hands work in a sterilized lead-covered box. The patient is mobilized within the first days following fixation. Crutches are used until roentgenographic evidence of bony union is present. The average age of these patients was seventy-three years, the average hospitalization twenty-four days, and the average operating time twenty-three minutes. There were no operative deaths and three operative failures.—*Richard McGovney, M.D., Santa Barbara, California.*

Sciatic Pain in Low-Back Derangements:

Its Incidence, Significance, and Treatment

A Symposium

PRESENTED AT THE ANNUAL MEETING OF THE

American Orthopaedic Association,

KANSAS CITY, MISSOURI,

on May 8, 1940

Introduction . . . . . *Fremont A. Chandler, M.D.*

Anatomical Variations and Roentgenographic Appearance of the Low Back in Relation to Sciatic Pain. *Theodore A. Willis, M.D.*

Neuro-Anatomical and Physiological Aspects and Significance of Sciatica . . . . . *Winchell McK. Craig, M.D. and Maurice N. Walsh, M.D.*

Conservative Treatment of Sciatic Pain in Low-Back Disability . . . . . *John G. Kuhns, M.D.*

Posterior Protrusion of the Lumbar Intervertebral Discs. *Joseph S. Barr, M.D. and William Jason Mixter, M.D.*

Spine Fusion for Protruding Intervertebral Discs. *Benjamin P. Farrell, M.D. and William B. MacCracken, M.D.*

Sacro-Iliac Conditions . . . . . *Marius N. Smith-Petersen, M.D.*

Low Backache and Sciatic Pain Associated with Spondylolisthesis and Protruded Intervertebral Disc: Incidence, Significance, and Treatment . . . *Henry W. Meyerding, M.D.*

Fasciotomy for Sciatic Pain . . . . . *Frank R. Ober, M.D.*

The Relief of Low-Back Pain and Sciatica by Release of Fascia and Muscle . . . . . *Clarence H. Heyman, M.D.*

The Fascial Elements in Associated Low-Back and Sciatic Pain . . . . . *Albert H. Freiberg, M.D.*

The Articular Facets in Relation to Low-Back Pain and Sciatic Radiation . . . . . *Carl E. Badgley, M.D.*

SCIATIC PAIN IN LOW-BACK DERANGEMENTS: ITS INCIDENCE, SIGNIFICANCE, AND TREATMENT

Introduction

BY FREMONT A. CHANDLER, M.D., CHICAGO, ILLINOIS, *Chairman*

In presenting a Symposium on "Sciatic Pain in Low-Back Derangements: Its Incidence, Significance, and Treatment", your officers and program committee have cooperated in selecting a subject which is wide in scope, is open to a multitude of interpretations in which fixed opinions of leaders of orthopaedic thought are in conflict, and is one that is slowly yielding to careful scientific study and analysis. The orthopaedic surgeon of wide experience, as represented by members of this Association, needs no review of the pitfalls of diagnosis or treatment. He readily recognizes the many potential etiological features of this symptom complex, and chooses from a wide variety of therapeutic measures in prescribing for his patient. As no panacea has been presented, the orthopaedic surgeon actively or passively follows the oscillations of the therapeutic pendulum, and, with the accumulation of experience, charts his own course of procedure in each case. Newer procedures are scrutinized, tested, and accepted or discarded.

The composite judgment of the membership of this Association expressed in a symposium on sciatic pain should constitute a balance wheel for all medical thought.

The breadth of the subject necessitates restriction of the range of discussion, so that a more thorough consideration of the orthopaedic phases may be developed. In order to focus attention on major divisions of the problem, your officers and committee have asked and have received the cooperation of the several essayists appearing on the program during the forenoon. These men have been asked to present their subjects with special relation to sciatic pain. Because of this limitation, their papers may not be as comprehensive as the titles would indicate.

During the afternoon, the symposium will be open for a thorough discussion. Several members have been requested to open discussion of the papers presented in the forenoon. They have been most cooperative in accepting these assignments.

To facilitate an orderly participation by as many members as possible, cards have been prepared for the use of those interested in taking part. The essayists will have twenty minutes for their presentations, the discussors five minutes.

ANATOMICAL VARIATIONS AND ROENTGENOGRAPHIC APPEARANCE OF THE LOW BACK IN RELATION TO SCIATIC PAIN

BY THEODORE A. WILLIS, M.D., CLEVELAND, OHIO

Except for a rare *Blitzkrieg* against a specific disease entity, progress of medical science has resulted from continuous pressure exerted on a wide front, advance salients being thrown out now here, now there, in the fields of bacteriology, parasitology, biochemistry, pathology, and physiology. In the excitement of victory undue importance has often been ascribed to local successes, and greater advance has been claimed than could be maintained.

In the campaign against low-back pain and sciatica one recalls the battles of Gynecology, Sacro-Iliac Subluxation, Postural Fault, Congenital Anomalies, Fascitis and Contractures, Spastic Pyriformis, Displaced Intervertebral Discs, Thickened Ligamenta Flava, and Narrowed Intervertebral Spaces. All of them were notable advances, but each of them, though temporarily overemphasized, was only a part of the campaign. Only time and perspective can attribute to each its relative importance in the general conflict.

The battle of Congenital Anomalies, which is to be discussed, like the others, overshot its true objective and was temporarily overpropagandized. That the reaction has not yet died out, nor the true importance of the advance been established, the writer is convinced; for one reason, because roentgenologist colleagues, who used to note in their reports all vertebral anomalies, have become negligent in this respect, and it was necessary for him to examine all the roentgenograms himself in collecting data for this discussion.

Theoretically, congenital anomalies of the lumbosacral and sacro-iliac areas of the skeleton may be of interest in the syndrome of low-back pain and sciatica either as predisposing or etiological agents, or as factors preventing recovery from these symptoms following injury. Certain anomalies weaken the anchorage of the spinal column to the pelvis. Others expose the contents of the neural foramina, particularly the last presacral nerve roots, to impingement, stress, or irritation from postural faults or disease.

It is unfortunate that the lumbosacral junction is the particular part of the human spinal column most subject to mechanical strain, and at the same time the part most often involved in anomalies and defects of development.

Two natural processes are to blame for this. First, an evolutionary shortening of the spinal columns of man and the other primates is being accomplished by inclusion in the sacrum of the last lumbar vertebra. Second, upright posture has been accomplished by hyperextension to

nearly a right angle at the lumbosacral area. The first process gives rise, in addition to numerical variation of presacral vertebrae, to the many anomalies resulting from partial sacralization of the last lumbar and partial lumbarization of the first sacral segments. These are evidenced as enlarged transverse processes articulating or impinging with the sacrum or ilia, narrow lumbosacral intervertebral spaces, sagittal-plane articular processes, and decrease in the anteroposterior diameter of the superior surface of the sacrum as compared to the inferior lumbar surface opposing it.

Lack of spinous processes, spina bifida, and interruptions in continuity of the neural arches, localized particularly at the lumbosacral level of the back, are examples of defective development of bone segments. Interruptions in the neural arch have been reported in all races of mankind, but not to the author's knowledge in human foetuses, or in the spinal column of any other vertebrate. This anomaly is, therefore, believed to be a consequence of assumption of upright posture.

The problem at hand is which, if any, of these anomalies and defects do, and which do not, play a part in the development of low-back pain with gluteal and sciatic distribution, or delay recovery from such symptoms arising from injury or disease. A brief discussion of these lesions and the ways in which they may so affect the body will be attempted.

It is doubtful whether the addition or subtraction from the spinal column of a normal presacral segment affects its stability to any appreciable extent. A longer back may be slightly more mobile, and a shorter one perhaps a little stronger.

It is doubtful whether bilateral, symmetrically enlarged transverse processes which do not impinge upon, or articulate with the sacrum or ilia weaken the sacro-iliac juncture. The claim has been made that these enlarged processes form a prominence over which the lower nerve roots are stretched, with resulting nerve irritation and sciatic pain. In a series of cases removal of these large transverse processes has been followed by relief of pain. Since processes and nerves develop simultaneously, it is improbable that they are maladjusted, but with development of lordosis or muscle spasticity the tension on the nerve or blood vessels might be increased, as in cervical-rib syndrome. Relief of sciatic pain has followed so many simple fasciotomies, however, that one is not justified in attributing the favorable postoperative reaction entirely to the removal of the enlarged transverse process.

It seems reasonable to suppose that asymmetrical anchorage of the lumbar column to the pelvis predisposes that part of the back to strains and sprains. The asymmetry may be in the size and attachment of the transverse processes or in the planes and inclinations of the articular facets. Variations of the latter particularly affect the direction and extent of lumbosacral mobility.

It is a fact that a congenitally narrow lumbosacral interspace approaching the first sacral interspace in depth, diminishes the size of the

foramina through which the last lumbar nerve roots emerge and thus increases liability of the transmitted nerves to pressure from accident, disease or lordosis. Differentiation of congenital from acquired narrowing of the intervertebral space must be difficult in most instances, but why a narrow space with increased overriding of the articular processes should be labelled backward displacement of the lumbar vertebra, or reversed spondylolisthesis, is a riddle. This diagnosis is based upon the relative position of the postero-inferior border of the last lumbar body to the posterosuperior border of the first sacral vertebra as shown by lateral roentgenograms, but the superior surface of the sacrum is so variable in form, its posterior border being either convex or concave toward the neural canal, that as a landmark it is most unreliable. This sacral surface was found of less anteroposterior diameter than the opposing lumbar in 66 per cent. of fifty skeletons examined.

The criticism, that museum material is worthless as evidence in proving the point, seems as preposterous as the basing of an opinion on the shadowgraph known as a roentgenogram without critical consideration of the parts casting the shadow.

It has been conclusively shown that in the great majority of vertebrae each lateral half of the neural arch develops from a single center of ossification. These two centers normally meet in the midline behind at about the time of birth, fuse, and develop the spinous processes. The lumbar spinous processes provide large surfaces for the attachment of the powerful ligaments and muscles that stabilize the lower back. Failure of the lower lumbar or the upper sacral spinous processes to develop therefore probably results in a less stable back. But sprain of a back is a matter of the strength of the affected individual in relation to the amount of force applied and his position when it is applied, and unfortunately the same individual cannot be tested with and without a spinous process.

As is well known, skeletal anomaly is often associated with defects of the soft tissues, particularly of nerve tissue. It is conceivable that cleft of the neural arch may well be associated with defective development of the spinal cord and its nerve roots in minor as well as major spina bifida. The author has been able to find very little information concerning neurological variations associated with the lesser degrees of spina bifida occulta. Dittrich reported a number of laminectomies done for sacral spina bifida occulta in all of which he found pathological fat, muscle, and fibrous tissue pressing or pulling on the nerve structures. Since these were found in muscular rheumatism, hematogenous osteomyelitis, and infantile paralysis as invariably as in spastic paralysis, eneuresis, dysmenorrhea, constipation, and the syndrome under discussion, one questions the importance of the pathology reported.

A lesion more definitely affecting stability of the lower back is the lateral defect of the neural arch. Hitchcock has come closest to explaining the etiology of this condition in attributing it to a fracture of one or both laminae incurred in early infancy before they are well ossified or

TABLE I

COMPARISON OF ANOMALIES FOUND IN THE PRESENT SERIES OF PATIENTS WITH THOSE IN PREVIOUSLY PUBLISHED STUDIES

Anomaly	Present Series 79 Cases		Museum Skeletons 748 Cases	Badgley's 447 Cases	Hodges' and Peck's 538 * Cases	Williams' 400 Cases
	No.	Per Cent.	Per Cent.	Per Cent.	Per Cent.	Per Cent.
No congenital anomaly.	44	55.7	88.0	72.9		
Partial lumbarization or sacralization.....	20	25.3	6.68	9.7	14.3	15.7
Narrow lumbosacral discs	6	7.6		57.2	12.8	71.25
Laminar defects.....	5	6.3	4.14‡	8.1**		3.25**
Spina bifida occulta.....	7	8.8	1.2†	10.8		
Asymmetrical facets....	11	13.9		21.9		2.0

* 353 cases had backache but no radiation.
† Last lumbar only.
** Spondylolisthesis.
‡ In another study of 1,520 skeletons, the author found 5.2 per cent. with laminar defects.

fused. The injured cartilaginous tissue fails to ossify and the anchorage of the spinal column to the sacrum depends upon fibrous union only. Hitchcock, Ferguson, and Friberg have each reported instances of this defect in which the consequent spondylolisthesis has occurred or progressed after injury.

Coexistence of the central and lateral defects of the neural arch would seem still further to affect the stability of the parts and their resistance to injury.

Muscle fatigue, in the lower back as elsewhere, gives rise to discomfort which increases as the cause of fatigue continues. As the musculature fails, more and more strain falls upon the ligaments and joint structures. These inelastic tissues gradually give way, but not without protest, and inflammatory changes occur, together with soreness, restricted mobility and pain both locally and along the distribution of the nerves that supply the affected parts.

Possible mechanisms of low-back and sciatic pain resulting from congenital skeletal anomalies are chronic muscle strain and ligament sprain, tension on, or compression of, nerve roots, and associated anomalies of nerve tissue.

Since there is no way of testing the strength and stability of a back with, and again without, the various anomalies, and since a surgical operation, or even such simple treatment as bed rest or postural exercise affects the part in different ways, proof of an etiological theory from the result of treatment is uncertain. Probably the best available evidence concerning their etiological importance is a determination of the incidence of the

various anomalies in a group of individuals suffering from low-back pain and sciatica and in a comparative group that is symptom-free.

In search of this information the records of the last 2,500 patients seen at the writer's office have been reviewed. Two hundred and twenty-nine, or 9.2 per cent., of them gave as their reasons for seeking professional advice low-back pain extending to one or both thighs or legs. It has been the writer's custom, if the etiology of the complaint seems clear and response to treatment is prompt, to spare the patient the expense of roentgenographic examination. After discarding these, and also those in whom there was proven pathology accounting for the complaint, for example: five with fascial contractures, twenty-two with arthritis, three with displaced intervertebral discs, four with ruptured and retracted ligamenta flava, three with tuberculosis, one with osteomalacea, and one with Paget's disease, there remained only seventy-nine patients or 34.5 per cent. in whose backs no definite pathological condition could be demonstrated.

Of these seventy-nine, twenty-three showed no skeletal anomalies. In twenty-one the transverse processes of the last lumbar vertebra were definitely large but made no contact with sacrum or ilia.

In twenty there was impingement, articulation, or fusion of one or both last lumbar transverse processes with the sacrum. The lumbosacral intervertebral space was definitely narrow in six. The neural arches were defective in five, in three of which spondylolisthesis had occurred. Spina bifida occulta was present in the last lumbar vertebra in two, and in the first sacral vertebra in five. Lumbosacral articular facets were in the sagittal plane in eleven,—four on the right, two on the left, and five bilateral.

Comparing these figures as far as possible with those obtained by the author from examination of 748 skeletons in the Hamann Museum, with Badgley's study of 447 patients who had low-back and sciatic pain, with the 538 control subjects of Hodges and Peck not presenting this syndrome, and with Williams' report of 400 patients with low-back and sciatic pain, Table I has been constructed.

It is found that only 56 per cent. of our small series were free from anomalies as compared to 72.9 per cent. of Badgley's. In the skeletal material only the transverse processes that actually impinged or articulated with the sacrum or ilium were recorded. There were 6.68 per cent. of these as compared with 25.3 per cent. in our series, 9.7 per cent. in Badgley's, 15.7 per cent. of Williams' and 14.3 per cent. of Hodges and Peck's controls.

The greatest divergence in the different series occurred in respect to the incidence of narrowing of intervertebral discs. The author found only 7.6 per cent. while Badgley found 57.2 per cent. and Williams 71.25 per cent. in their patients with backache. Hodges and Peck found 12.8 per cent. in their control patients. The author cannot account for this great difference. Badgley, and Hodges and Peck classified a disc as narrow if it were not more than one-half the thickness of the next preced-

ing one. Williams did not state the criterion upon which he based his diagnosis of narrowing. It was impossible of course to obtain this data from skeletal material, but Todd and Pyle, in measuring the discs in a series of cadavera found that the lumbosacral discs averaged about two millimeters more at the anterior borders than the discs next above.

The lateral neural arch defect was diagnosed about 1 to 3 per cent. more frequently in painful backs than in the dissecting-room material, except that in Williams' group of patients with painful backs it was diagnosed in 2 per cent. fewer. Diagnosis of this defect in a living patient is difficult and is much more apt to be overlooked than in a macerated skeleton.

Spina bifida occulta was present in seven or 8.8 per cent. of the author's patients, two in the last lumbar and five in the first sacral segments; and in 10.8 per cent. of those of Badgley. In the skeletal subjects it was found in the last lumbar in 1.2 per cent. Its incidence in the first sacral was not noted. This defect is easily diagnosed and the difference of incidence in skeletons, and normal and painful backs is apparently not great.

In eleven, or 13.9 per cent. of this series, and 21.9 per cent. of Badgley's, the lumbosacral articular facets were in the sagittal plane or otherwise asymmetrical. Unfortunately these were not included in the classification of skeleton variations.

There are certain discrepancies in the classifications of these groups of patients that affect their comparison. Badgley included in his group patients in whom there were pathological lesions to account for the symptoms. Hodges and Peck state that of their 538 controls 353 had backache but no referred pain. It is of course possible that the referred pain had merely not yet developed. Inclusion of these individuals (65 per cent. of their controls) affects their data to start with. Also an indefinite number of the skeletal museum subjects may have had backache with referred pain while alive.

Allowing for these differences in classification, there is still a greater incidence of anomalies in painful backs than in those free from symptoms or in the run of dissecting-room cadavera. The greatest variation in the respective series is in the incidence of narrow lumbosacral intervertebral discs, but since those of congenital origin cannot, or at least have not, been differentiated from those of pathological origin, an intelligent appraisal of their etiological relation to backache and referred pain is difficult.

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NEURO-ANATOMICAL AND PHYSIOLOGICAL ASPECTS AND SIGNIFICANCE OF SCIATICA

BY WINCHELL McK. CRAIG, M.D., AND MAURICE N. WALSH, M.D.,
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Sciatica, an inclusive term which is used to describe a painful discomfort along the posterior aspect of the hip, thigh, and leg, has since ancient times baffled physicians in the matter of diagnosis and treatment. The numerous methods of treatment which have been heralded down through the ages as successful testify to the difficulties encountered in finding a therapeutic agent for this condition, which is permanent in effect. One of the reasons for such difficulties is the fact that the sciatic nerve is the longest nerve in the body, and, therefore, subjected to trauma and infection throughout its entire length.

The anatomical development of the lower extremities and the innervation of them was described by Stookey as follows:

"The morphological segmental arrangement, such as is found in the trunk musculature, is replaced in the extremities by a pleurisegmental arrangement, so that each muscle is innervated by nerves from several segments, thus necessitating the development of plexuses. . . .

"The formation of the limb plexuses and the distribution of their branches may be best understood by considering the development of the extremities, for in the development is found the explanation of the division of the spinal nerves into ventral and dorsal branches and, in general, their ultimate muscular distribution.

"In the human embryo the limb buds appear at about the third week. The cephalad pair lie opposite the lower four cervical and first thoracic vertebræ and the caudad pair opposite the lower lumbar and the first sacral. . . . Only the ventral primary division of the spinal nerves enter into the innervation of the limb bud approximately five for the upper extremity and seven for the lower, thus indicating that the limb buds are the outgrowth from a number of segments and are more closely associated with the ventral than with the dorsal surface of the body, since the ventral and not the dorsal primary divisions of the spinal nerves are associated with them. . . ."

"In the development of the lower extremity torsion and rotation of the femur, . . . take place . . . so that its preaxial border is turned medial and cephalad, its postaxial, lateral and caudad. Thus the dorsal or extensor musculature comes to lie ventral, and the ventral or flexor musculature, dorsal. . . . From an embryological standpoint it follows then that the primitive dorsal musculature, though rotated ventrally, should be innervated by the dorsal divisions of the lumbosacral plexus, and the

primitive ventral musculature, though rotated dorsally, by the ventral divisions."

Woltman has stressed the point that almost no type of pain is pathognomonic, but that a given disease may be accompanied by many kinds of pain, and that, although pain may be the only symptom manifested, much may be learned by the physician if he will inquire about the mode of onset, duration, and intensity of the pain. Pain is a subjective complaint, something that cannot be seen and cannot be palpated, and necessitates cautious interpretation of what the patient has to tell.

Pain, occurring in the distribution of the sciatic nerve, may be caused by a lesion of the spinal cord or nerve roots (especially from the fourth lumbar to the second and third sacral segments from which the sciatic nerve and its components are derived), by affections of the lumbosacral plexus, or by lesions of the sciatic nerve itself.

Pain which is more or less limited to the sciatic distribution has been observed to accompany lesions of the lumbar, thoracic, or even cervical portions of the spinal cord.

Pain caused by intramedullary lesions of the spinal cord usually can be distinguished from that caused by irritation of the posterior nerve roots and ganglia. The so-called central pains of this type are usually produced by irritation of the spinothalamic pathways. Central pain caused by lesions of the thalamus usually is not difficult to distinguish from that caused by lesions of the cord and is not definitely sciatic in nature. Such pain is rare. It is of a peculiar, irritating nature, and the patient is often unable to localize the pain accurately although it is usually felt in the arm and leg opposite the lesion. Such pain is often accompanied by dysaesthesia of a burning, boring nature. Head and Holmes believe that such pain is caused by the removal of the inhibitory control which the cerebral cortex normally exerts on the subcortical centers concerned with the perception of this form of sensation. Pain of central origin referable to lesions of the spinal cord is uncommon. When it occurs, however, it may be of a peculiar, agonizing type which is sometimes described as "aching, breaking, or piercing" and which is accompanied by dysaesthesia and various disagreeable sensations such as a sense of "burning" elicited by pinpricking and "vibratory electric-light sensation" produced by thermal or tactile stimuli. There is usually marked loss in the various forms of sensation below the level of the lesion, together with hyperactivity of the tendon reflexes with the occurrence of pyramidal tract signs below the level of the lesion. In the cervical or lumbar region there is a loss of tendon reflexes if the lesion occurs in the segments which mediate tendon reflexes from the extremities, such as the cervical or lumbar enlargements of the spinal cord. The pain of central lesions usually is not affected by coughing and sneezing and is rarely worse at night. There may be local tenderness to percussion at the site of the lesion.

Since a large number of patients suffering from sciatic pain have lesions involving the nerve roots which compose the cauda equina, an accurate knowledge of the anatomical aspects of this structure is essential.

Anatomically, the spinal cord usually terminates opposite the intervertebral disc below the first lumbar vertebra. The nerve roots forming the cauda equina (Fig. 1) consist of those which arise from the lumbar and sacral regions of the spinal cord, descend in the subarachnoid space intradurally, and are freely movable except as they approach their exits, where they are fixed. Thus it is that intraspinal lesions can displace and impinge on nerve roots without causing any motor, sensory, or reflex changes, and can produce the objective symptom of pain. It is a well-known fact that the pain of caudal tumors masquerades as low-back and sciatic pain for months and years before the tumor produces any objective neurological signs, and for the same reason protruded intervertebral discs and hypertrophied ligaments can produce pressure on nerve roots without producing any symptom except pain.

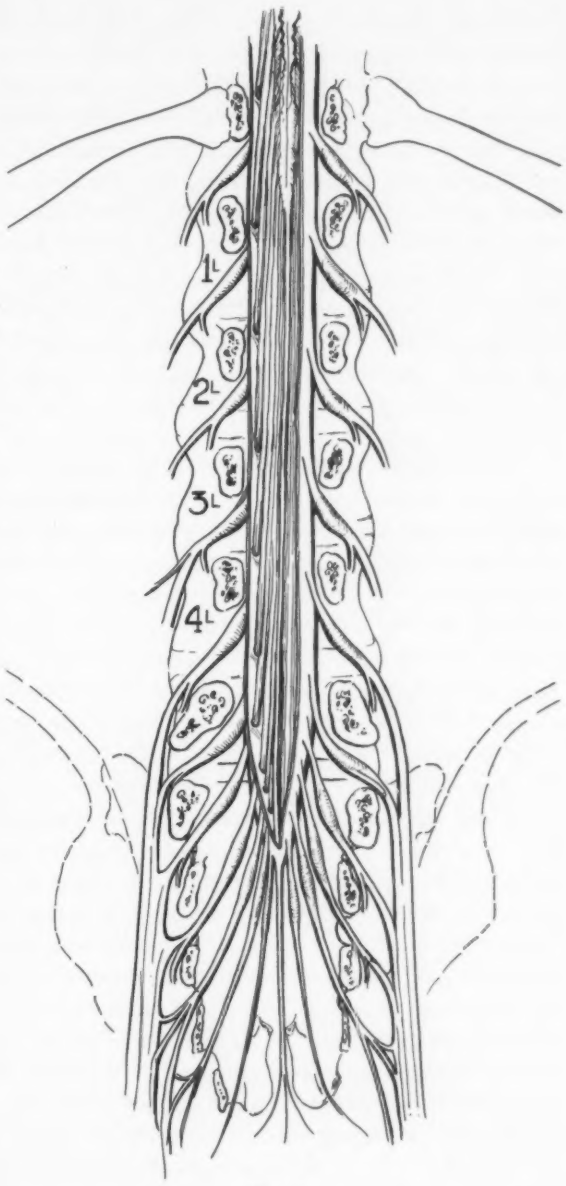

FIG. 1

Anatomical aspects of the lumbosacral region of the spinal cord with the dura open to show the relationships of the nerves to the conus medullaris.

Intraspinal lesions of the fourth and fifth lumbar or the first and second sacral nerve roots immediately after their emergence from the cord, during their course as part of the cauda equina, or in the neighborhood of the intervertebral foramina, commonly produce sciatic pain. Lesions involving or compressing these nerve roots give rise to the type of pain

known as "radicular pain". This type of pain usually is sharp, rarely aching or burning, and is often exacerbated by coughing, sneezing, or sharp flexion of the head on the chest. It tends to be worse at night, a fact which is presumably due to elongation of the spinal column while the patient is in the supine position, with resulting traction on the nerve roots. It is commonly accompanied by local tenderness to percussion over the region of the spinal column at the level of the lesion, muscular weakness, reflex changes or paraesthesia. Sensory changes, should more than one nerve root be involved, may be found, the dermatome or dermatomes affected depending on the nerve roots which are compressed. It should be emphasized, however, that all the above features need not be present to permit the diagnosis of radicular pain. In fact, it is rather rare for one patient suffering from radicular pain to demonstrate all these characteristics.

Since the spinal cord comes to an end opposite the intervertebral disc of the first lumbar vertebra, the lumbar and sacral nerve roots which compose the sciatic nerve arise opposite the twelfth thoracic and first lumbar vertebrae. Thus, extramedullary lesions at this level may produce sciatic pain of radicular type, and this is probably the highest level at which it can be obtained. Lesions at this level, however, are very likely early to involve the centers for control of the sphincter muscles, since the parasympathetic nerve supply for the urinary bladder and sphincters arises from the second and third sacral segments of the cord and the sympathetic nerve supply arises from the second to fifth lumbar segments, whereas the first to the third sacral segments mediate the control of the rectal sphincters, as well as the genital muscular system.

The recent work of Suh and Alexander on the blood supply of the spinal cord tends to indicate that the lumbar radicular vessels are important sources of blood supply for the major part of the spinal cord, whereas the middle thoracic section of the spinal cord has the poorest segmental circulation. This is of interest, since myelitic processes are more common in the latter situation. Suh and Alexander wrote, "the blood supply of the human spinal cord depends on from six to eight anterior radicular arteries, from five to eight posterior radicular arteries, from six to eleven anterior radicular veins and from five to ten posterior radicular veins, the largest always being an artery and vein in the lumbar region, the arteria and the vena radicularis magna." The arterial supply for the lumbar part of the spinal cord usually is accompanied by the second and third lumbar nerve roots, and the regions of the cord, dorsal and ventral to this, are supplied by these arterial vessels.

Because of the anatomical relationships, lesions of the nerve roots comprising the cauda equina offer special problems in diagnosis. Those lesions involving the spinal cord itself tend to produce sharply localized segmental sensory, motor, and reflex changes. This is frequently not the case when lesions involving the nerve roots in the cauda equina are present. The roots are freely movable and small lesions in the spinal canal may

cause no symptoms. The lesion may involve several nerve roots, a circumstance which confuses accurate localization. The pleurisegmental nerve supply to the muscles of the lower extremities and the tendon reflexes mediated by more than one segment of the spinal cord increase the difficulty.

One of the basic concepts in the physiology of the brain and spinal cord is that the central nervous system is organized in terms of movement patterns rather than in anatomical segments. Sherrington [20] wrote "The simplest spinal reflex, as Hughlings Jackson was wont to insist, 'thinks,' . . . in movements, not in muscles." Thus, when the foot of an animal whose brain has been destroyed is pinched, the resulting reflex is not restricted to muscles of the dermatome involved; rather, a withdrawal of the whole limb occurs. Theoretically, a segmental spinal reflex involves two elements, an afferent neuron and a motor unit. In most cases, however, the connections of the afferent neurons are such that each may excite motor units of several spinal segments through so-called internuncial neurons, to produce patterns of movement rather than contractions of specific muscles. The patellar reflex involves the second, third, and fourth lumbar nerves, and the corresponding segments in the spinal cord; the Achilles and internal hamstring reflexes, the fourth and fifth lumbar, and the first and second sacral nerves and segments, whereas the external hamstring reflex is mediated by the first, second, and third sacral nerves and segments.

The distribution in the skin of the spinal nerves forms well-defined, although overlapping, areas known as "dermatomes". Sherrington [21] pointed out that the field of each sensory root overlaps those of adjacent roots, and that section of a single root does not result in any degree of anaesthesia. He also pointed out that although in the plexuses associated with the innervation of the extremities each segmental nerve contributes sensory fibers to two or more peripheral nerves, the cutaneous distribution of these fibers is not composed of disjointed patches but forms a continuous field.

Foerster, working with patients, was able to verify and extend the work of Sherrington. He defined the dermatomes by outlining the borders of the sensibility remaining after a large number of contiguous posterior nerve roots were divided and after a single root in the middle was left intact. In addition, faradic stimulation of a distal part of a divided posterior nerve root was carried out, producing vasodilatation in the corresponding dermatome. Foerster and others believe such vasodilatation is due to the fact that the posterior roots carry efferent fibers and that stimulation of these fibers causes vasodilatation.

Foerster demonstrated that no regions of anaesthesia are found unless two or more segments are physiologically incapacitated. He further showed that lesions producing isolated segmental anaesthesia with intact segments above and below are rare, but that when they are present, they help to localize the lesion.

Therefore, it is readily seen why accurate localization of lesions in the cauda equina is extremely difficult and even, in many cases, impossible; especially is this true when posterior protrusions of the intervertebral discs produce the clinical picture of tumors of the cauda equina. At present, contrast media are usually employed to localize the lesion accurately. As knowledge of the syndrome has increased, however, it has been possible to diagnose and localize more or less accurately certain instances of protrusion of interve tebral discs, so that direct exploration is possible. Because more than 90 per cent. of instances of protruded discs occur at the fourth and fifth lumbar interspaces, it should be possible to predict the situation of the lesion among patients who have the typical syndrome of protruded intervertebral disc. Spurling and Grantham have recently stated that they were able to localize accurately posterior protrusion of a disc in the lumbar region to the exact intervertebral space in fully 50 per cent. of cases.* They give the following criteria for localization of the protrusions to the third, fourth, and fifth interspaces: At the third lumbar interspace there was found "disability of the lower part of the back, with local tenderness at the third lumbar spine and reduction of lumbar lordosis," associated with "positive Lasègue test" and "positive Naffziger test, producing paraesthesias in the fourth and fifth lumbar dermatomes". There was "reduction or absence of the knee jerk; ankle jerk unchanged", and "hypaesthesia and paraesthesias in the fourth and fifth lumbar dermatomes". At the fourth lumbar interspace there was "disability of the lower part of the back with stiffness of the lumbar portion of the spine and localized tenderness at the level of the fourth lamina, with reduction of lumbar lordosis" and "positive Lasègue test" with "positive Naffziger test with paraesthesias involving the fifth lumbar, the first sacral and perhaps the second sacral dermatomes". The ankle and knee jerks were uninvolved. "Hypaesthesia and paraesthesias [were found in the fifth lumbar and first sacral dermatomes." At the fifth lumbar interspace there was "disability of the lower part of the back with absence of lumbar lordosis and localized tenderness to pressure over the fifth lumbar vertebrae", and "positive Lasègue test" as well as "positive Naffziger test, producing paraesthesias radiating into the first and second sacral dermatomes". There was diminution or absence of ankle jerk and "hypaesthesia involving the first and second sacral dermatomes".

However, Spurling and Grantham had only one patient suffering from a protrusion ("ruptured intervertebral disk with herniation of the nucleus pulposus") at the third interspace; thirty-nine with protrusions at the fourth and fifty-one, at the fifth interspaces. The authors' experience does not coincide with Spurling's in this regard. Their results of a

* It may be noted here that Spurling and Grantham prefer the term "rupture of the intervertebral disk" to "protruded intervertebral disk." Their explanation of such a preference is: "In order for the disk to produce symptoms we believe the annulus fibrosus must have been ruptured or torn, and, if the nucleus pulposus is extruded through the tear, a certain group of symptoms will usually appear; if the nucleus is not so extruded, however, another set of symptoms will be encountered."

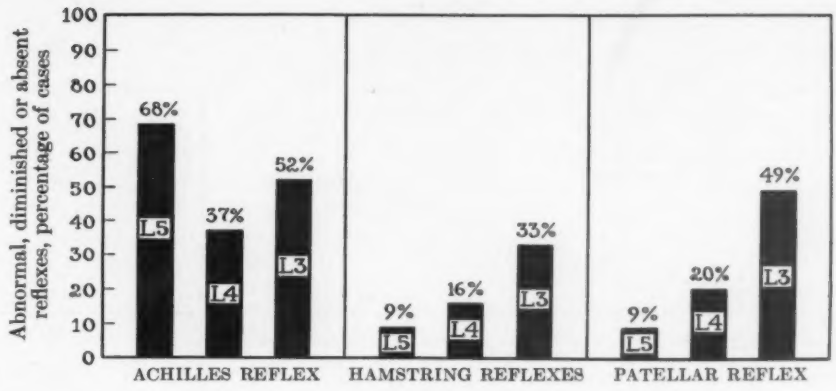

FIG. 2

Results of tests for the presence or absence of reflexes, and also for diminished reflexes, in 449 instances of protrusion of a single intervertebral disc.

careful study of the reflexes in 449 protrusions of single discs in the lumbar region (out of a complete series of 500 protrusions, both single and multiple) may be found in Figure 2.

It will be noticed that in 68 per cent. of patients with protrusions at the fifth interspace, 37 per cent. of those with protrusions at the fourth interspace, and 52 per cent. of those with protrusions at the third interspace, the Achilles reflex was either absent or diminished. Similarly confusing observations have been noted concerning the hamstring and patellar reflexes. Here it was found that 21 per cent. of the protrusions of discs at the fifth interspace, 57 per cent. of those at the fourth interspace, and 28 per cent. of those at the third interspace are accompanied by no reflex or sensory findings whatever. (These latter figures are not depicted in this study.) It is thus obvious that diminution of the Achilles reflex is more common in protrusions of the fifth interspace, and that the patellar reflex is more frequently involved in protrusions occurring at the third interspace. However, in view of the large number of patients whose condition does not fit into the scheme proposed by Spurling, its value for practical diagnosis may be questioned.

It is important to remember that involvement of one nerve root alone cannot be expected to produce sensory changes, and that consequently, only those lesions which involve two or more nerve roots will produce a loss of sensation. Spurling stated, however, that paraesthesia may be the result of involvement of one nerve root alone. In the authors' entire series of 500 cases of both single and multiple protrusions of the intervertebral disc, 50 per cent. of the patients noted paraesthesia, but only 22 per cent. had sensory loss. Careful study of the dermatomes involved in the sensory loss demonstrated that there was little if any difference between protrusions of single discs at the third, fourth, and fifth interspaces, with the fifth lumbar and first sacral, the most commonly involved dermatomes.

Jung and Brunschwig, and others reported no evidence of sensory

TABLE 1

COMPARATIVE SYMPTOMS IN 285 CASES* OF PROTRUDED INTERVERTEBRAL DISC AND 154 CASES OF INTRASPINAL TUMOR, FOR DIFFERENTIAL DIAGNOSIS

Symptoms	Protruded Intervertebral Disc (285 Cases)		Intraspinal Tumor (154 Cases)	
	Cases	Per cent.	Cases	Per cent.
Unilateral sciatic pain	215	75	58	38
Bilateral sciatic pain	42	15	36	23
Low-back pain	271	95	65	42
Nocturnal pain	61	21	73	47
Sphincteric incontinence	14	5	51	33
Lasègue's sign present	231	81	22	14
Achilles reflex diminished or absent	171	60	36	23
Hamstring reflexes diminished or absent	51	18	42	27
Muscle paresis	63	22	65	42
Muscle atrophy	5	2	39	25
Sensory loss	62	22	98	64

* These 285 cases represent those in which sensory changes were present. They are part of the complete series of 500 cases of single and multiple protrusion of the intervertebral disc in the lumbar region, mentioned in the text.

nerve supply to the intervertebral disc, although the posterior longitudinal ligament was well supplied with sensory nerves. The innervation of the annulus fibrosus and the posterior longitudinal ligament has been recently restudied carefully by Roofe, who found that each of the spinal nerves gives rise to a recurrent branch just distal to the posterior root ganglion which reenters the intervertebral foramen and supplies the ligamentous structures two vertebrae lower than the exit of the spinal nerve (Fig. 3-A). The profuse supply of sensory nerve endings in the annulus fibrosus and the posterior longitudinal ligament may account for some of the heretofore unexplained painful symptoms in cases in which the compression is limited to the lower lumbar and lumbosacral regions, and in which a tear of the annulus fibrosus has led to herniation of the intervertebral disc. It is possible that this observation also may explain the occasional puzzling observation of sensory changes in a dermatome higher than the level at which protrusion of the disc or the tumor found at operation occurred.

At the fourth intervertebral space, the fifth lumbar nerve root lies in the subarachnoid space fixed laterally against the dura, from which it emerges below the level of the fourth lumbar interspace. It passes out

beneath the pedicle of the fifth lumbar vertebra through the intervertebral foramen. All the lumbar nerve roots and their respective discs, except the first sacral nerve which separates from the dural sac above the lumbosacral disc, bear this same relationship. Thus it might be expected that posterior protrusion of the fourth intervertebral disc most commonly might involve the fifth nerve root, whereas protrusion of the fifth interspace might involve the first sacral nerve root. However, in this series of cases it has been observed not infrequently that lateral protrusion of the fifth interspace may compress the fifth nerve root, and those disc protrusions at the fourth interspace, the fourth nerve root; whereas a large posterior protrusion of a disc, particularly a midline protrusion, may compress several or even all nerve roots in the cauda equina, confusing any attempt at accurate localization of the lesion by clinical means alone.

A differential diagnosis between a tumor of the cauda equina and protruded intervertebral disc is sometimes very difficult. The marked tendency toward intermittency of symptoms (84 per cent. in the entire series of 500 cases) caused by protruded discs, as contrasted with tumors in this region of which the symptoms are more often steadily progressive, may be confusing, especially when coupled with the fact that 26 per cent. of the patients in the series had no reflex or sensory changes. In comparing the clinical changes occurring in cases of protruded intervertebral disc and intraspinal tumor, only 285 cases were analyzed in the complete series of 500 cases of single and multiple protrusion of intervertebral discs (Table I). These 285 are cases in which sensory changes occurred. The symptoms they produced are compared in Table I with symptoms produced in 154 cases of intraspinal tumor. In view of the present increased knowledge of the syndrome of protruded disc, however, it would be possible to explore the fourth and fifth lumbar interspaces directly, and avoid the use of a contrast medium, but the investigator should always keep in mind the fact that posterior protrusions of discs may occur rarely in the third, second, or first interspace, that multiple protrusion of discs may occur, and that removal of only part of the cause for the patient's symptoms cannot be expected to give symptomatic relief (Table I).

Patients who have low-back and sciatic pain may have no demonstrable lesion involving the intraspinal structures, the nerve roots, or peripheral nerves. Certain of these patients will have lesions so small and undeveloped that objective evidence of their presence or a clinical picture typical enough to permit their diagnosis on the basis of the history alone will be lacking. In addition to these, there is another group of patients who complain of sciatic pain and pain in the buttock, leg, and thigh, but in whom no organic cause for such pain is apparent. Such pain has been accepted as being referred from some adjacent or even remote pathological process.

Head wrote, ". . . where a painful stimulus is applied to a part of low sensibility in close central connection with a part of much greater sensibility the pain produced is felt in the part of higher sensibility rather

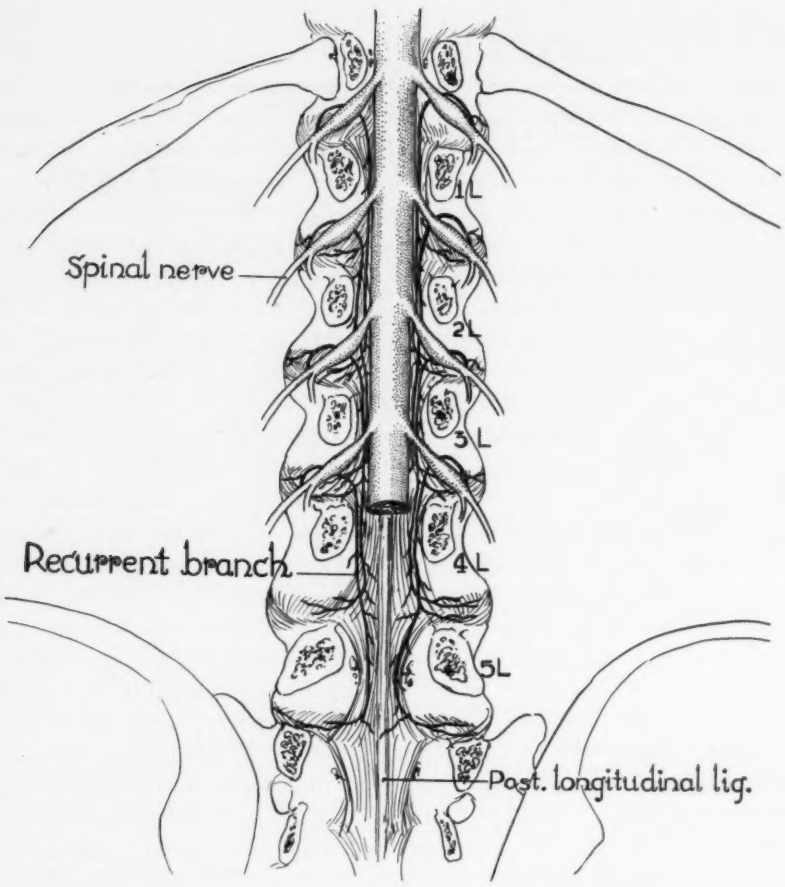

FIG. 3-A

Innervation of the intraspinal ligaments (after Spurling and Roofe).

than in the part of lower sensibility to which the stimulus was actually applied".

Referred pain is of common occurrence in the presence of intrathoracic and intra-abdominal disease,—for example, the pain felt in the left arm caused by coronary occlusion, the pain referred to the angle of the left scapula and produced by irritation of the center of the diaphragm, and the referred pain felt in the abdominal wall, referable to irritation of the periphery of the diaphragm. The explanation for such referred pain is not entirely satisfactory.

It was thought by Mackenzie that sensory manifestations of visceral disease localized in somatic regions are reflex in nature. These, he designated "viscerosensory" reflexes and explained them on the basis of hyperirritability in the corresponding segments of the spinal cord referable to exaggerated visceral stimulation. This irritation of a visceral organ sets up an exaggerated flow of nerve impulses which enter the correspond-

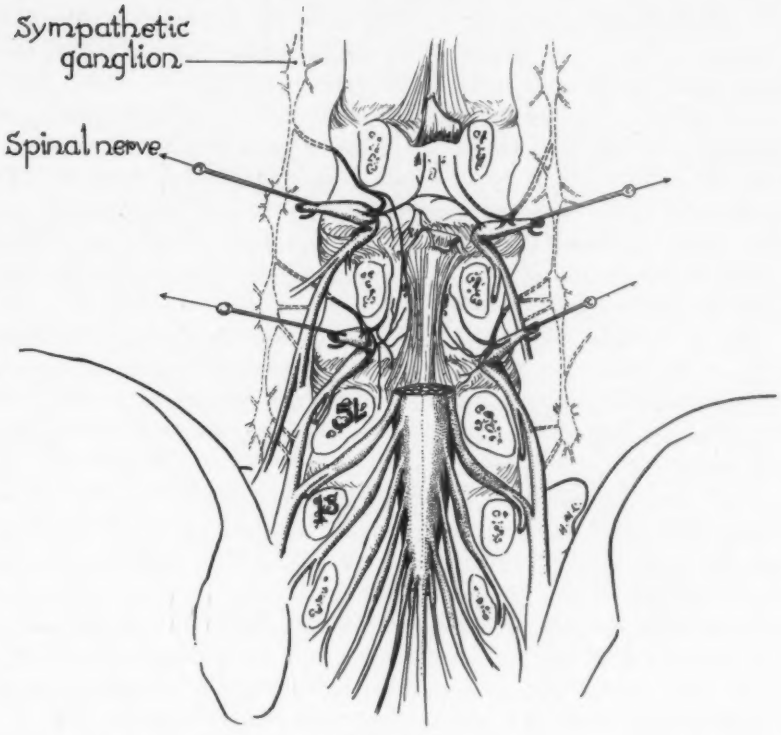

FIG. 3-B

Sympathetic innervation of the intraspinal ligaments (after Toldt).

ing segments of the spinal cord. These in turn give rise to an "irritable focus" in which the threshold of stimulation is so reduced that the normal impulses arising in the skin, muscles, and other peripheral structures produce painful sensations referred to the periphery in the somatic segments in question. Both the somatic pain or hyperalgesia and the muscular rigidity associated with visceral disease, according to Mackenzie's theory, are expressions of hyperirritability in the spinal cord, the pain or hyperalgesia being produced by exaggerated viscerosensory reflexes; the muscular rigidity, by exaggerated visceromotor reflexes.

Szemzo advanced the theory that visceral afferent fibers, in part, terminate in the posterior cell column in the spinal cord in neurons related to the spinothalamic system, and that the hyperirritability of these cells, by reason of excessive visceral stimulation, explains the phenomenon of somatic hyperalgesia in the presence of visceral disease.

The importance of impulses arising at the periphery in the phenomenon of referred pain is emphasized by the results of clinical and experimental study reported by Weiss and Davis. In twenty-five patients who experienced well-marked and definitely localized referred pains caused by visceral diseases, including angina pectoris, pleuritis (pneumonia), carcinoma of the oesophagus, gastric ulcer, cholecystitis, nephrolithiasis,

acute appendicitis, salpingitis, and pyelitis, the pain was either greatly alleviated or completely abolished by infiltration of the painful cutaneous regions with a 2-per-cent. solution of procaine hydrochloride. Referred pain induced experimentally in two normal human subjects by distention of a segment of the duodenum or the lower portion of the oesophagus by means of a rubber balloon was abolished in the same manner. These observations have been confirmed by Morley, and Rudolf and Smith. Woollard and Roberts, on the other hand, failed to abolish referred pain induced by irritation of the diaphragm, when they anaesthetized the corresponding cutaneous region.

Weiss and Davis explained their results on the basis of Mackenzie's theory of referred pain,—for example, segments of the spinal cord were rendered hyperirritable by abnormal visceral stimulation, and the normal afferent impulses from the skin entering these segments of the spinal cord gave rise to pain. The cutaneous region in which the referred pain is localized does not include the entire region supplied by afferent fibers entering the spinal segments which, according to Mackenzie's theory, are supposed to be hyperirritable. In view of this, the abolition of referred pain by means of anaesthesia of the localized cutaneous region can be explained more satisfactorily on the assumption that the pain is caused by overstimulation of the peripheral receptors by conditions produced reflexly at the periphery, the afferent impulses entering the spinal cord from the diseased or irritated viscus or the tissue adjacent to it. This view is supported by Davis and Pollock, who found that efferent stimulation of the sympathetic nerves extending into the head gave rise to pain by reason of the effect of the peripheral response to such stimulation on sensory end organs (Fig. 3-B).

The reflex arcs, through which the peripheral vasomotor and pilomotor reactions are mediated, comprise a general visceral afferent or a somatic afferent neuron, through which afferent impulses are conducted from the site of the lesion, or the irritable tissue adjacent to it, to the spinal cord. A visceral efferent neuron, situated in the intermediolateral cell column, and a sympathetic neuron, situated in the corresponding ganglion of the sympathetic trunk, conduct efferent impulses to the smooth muscle in question. In so far as cutaneous impulses are a factor in the production of referred pain, they are conducted by the pain-conducting fibers distributed to the skin and cutaneous blood vessels.

That all the nerve reactions involved in the referred somatic manifestations of visceral disease are facilitated by the hyperirritability of the centers in the spinal cord produced by the exaggerated visceral stimulation seems probable. The degree of hyperirritability produced in the spinal cord, furthermore, may be a determining factor in the intensity of both the direct visceral pain and the referred pain.

Since the joints, ligaments, muscles, and tendons of the lower part of the back are subjected to unusual strain, a study of their innervation has been made with the idea of attempting to learn in which region pain

referred from lesions of these structures might be expected to occur. Pitkin and Pheasant pointed out that dissections by Rüdinger and Smith-Petersen demonstrated the nerve supply of the sacro-iliac and sacrolumbar articulations and their accessory ligaments, and concluded that referred pain from these regions, spoken of by them as "telalgia", affects the gluteal or sacral region (or both) and that it may affect any part or all parts of the lower extremities and genito-inguinal regions except the internal crural and plantar regions.

Badgley emphasized the possibility that low-back pain with extension into the leg might be caused by a mechanism essentially produced by reference from a primary lesion, situated in the lumbar, lumbosacral, or sacro-iliac regions which was muscular, articular, or skeletal in origin.

Steindler called attention to the fact that spinal nerves are grouped into anterior and posterior primary divisions. The anterior divisions make up largely the cervical and lumbosacral plexuses, and lesions affecting these anterior divisions give rise to the well-known syndrome of radicular pain. He wrote of patients suffering from this type of referred pain as having the "posterior syndrome". It is diagnosed by the finding of tender points over the site of the lesion. Injection of these tender points with procaine hydrochloride, according to Steindler, produced important diagnostic information.

According to Steindler, Purkinje and von Luschka described the nervus sinuvertebralis which receives a white ramus from the common trunk and a gray ramus from the sympathetic chain just outside the intervertebral foramina and which turns back into the intervertebral canal, supplying the neck and head of the rib, the pedicles and the interior of the bodies of the vertebrae, the loose areolar perimeningeal tissue, and the periosteum. Von Luschka believed that they transmit the sensation of spinal irritation, paraesthesia, chilling, and sensations of heat from the spinal column. Steindler has suggested, on this basis, that reflex pain may be referable to lesions situated in the intervertebral canal rather than to direct compression of nerve roots.

Ober has emphasized the possible contracture of the fascia lata in the causation of sciatic pain, and has recommended fasciotomy for its relief. He did not mention, however, any mechanism by means of which it was produced, although it has been suggested that in this syndrome there is a referred pain of the type mentioned previously.

Macey has recently called attention to the rôle of disease of the intraspinous ligaments in the production of low-back pain, and definite disease of the intraspinous ligament has been demonstrated on biopsy. Relief of the pain which follows injection of procaine hydrochloride into the involved ligaments has been utilized as a diagnostic sign.

With these and a few other exceptions, however, pathological proof of lesions situated in the fascia, muscles, and joints usually is lacking. It is extremely probable that referred pains do occur in the lower extremity, but it is difficult to estimate their frequency in the light of present knowl-

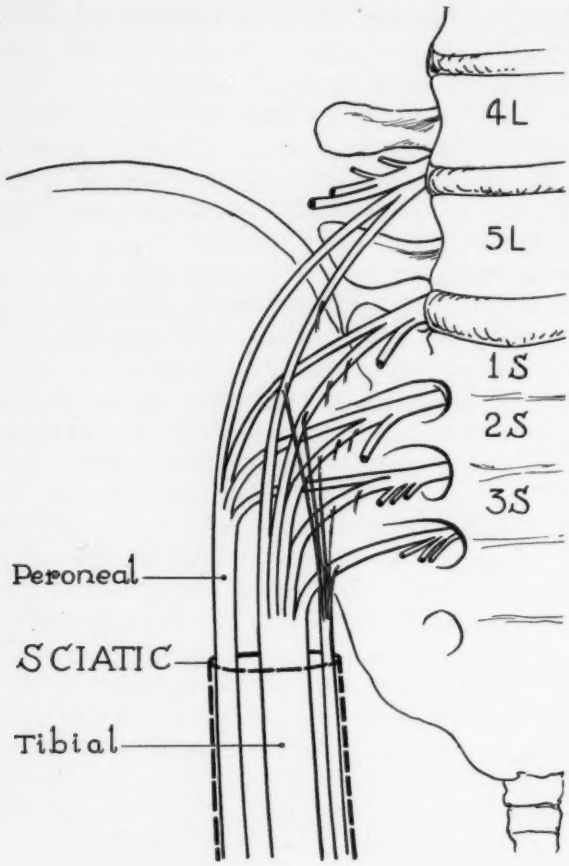

FIG. 4

Lumbosacral nerves from which the sciatic nerve is derived.

edge. An intraspinal lesion should be eliminated before the physician concludes that the patient's pain is referred from some distant focus. The structures of the lower part of the back probably are subjected to more stress and strain than those of any other region of the human spinal column. However, if pain referred from ligaments and joints of the spinal column were of extreme frequency, it might be expected that such pain would occur in the cervical and thoracic regions, and that it would be referred down the nerves of the arms and around the thorax and abdominal walls. As a matter of fact, in our experience the production of pain of this type in the arms, thorax, and abdomen, which is sufficiently localized to be confused with pain caused by radicular irritation in these regions, is extremely rare if it occurs at all.

Lesions of the nerve roots in the intervertebral foramina are relatively uncommon. Involvement of these structures by fracture of vertebral laminae, by hypertrophic osteitis of the laminae, or by marked spondylolisthesis rarely occurs. It should be emphasized, however, that the intervertebral foramina through which the nerve roots pass in leaving the spinal cord are, under most circumstances, amply large for their passage. Diagnosis of such lesions as those mentioned in this paragraph, however, is dependent on roentgenographic demonstration of the bone lesion plus neurological evidence of involvement of one or more nerve roots.

The occurrence of referred pain in patients who have pathological conditions of the intervertebral articulations has been explained by Steindler by calling attention to the nervus sinuvertebralis described by Purkinje in 1845 and by von Luschka in 1850.

After leaving the intervertebral foramina, the lumbar, sacral, and coccygeal nerves, in common with most spinal nerves, are grouped into anterior and posterior primary divisions. The lumbosacral plexus is formed by the union of the anterior primary divisions of the lumbar, sacral, and coccygeal nerves. In their relationship to sciatic pain, the lumbar and sacral plexuses will be considered separately. It should be added that sciatic pain is rarely produced by lesions of the lumbar plexus; it is, however, not infrequently produced by lesions of the sacral plexus. The nerves which supply the lower extremity have their origin in the lumbosacral plexus. The components are distributed to the lower extremities in a manner homologous and similar to the distribution of the brachial plexus to the upper extremities; the lumbar nerves are distributed similarly to the nerves formed from the anterior (medial and lateral) cords of the brachial plexus, and the sacral nerves are distributed in a manner similar to the distribution of the nerves from the posterior cord of the brachial plexus.

The anterior primary divisions of the first three lumbar nerves and the greater part of the fourth lumbar nerve form the lumbar plexus, which is situated on the posterior part of the psoas major muscle in front of the transverse processes of the lumbar vertebrae. Because of its situation, the lumbar plexus may be involved by tumors of the vertebrae or the retroperitoneal lymph nodes, psoas abscesses, or bullet wounds. Lesions of the lumbar plexus may be diagnosed by involvement of the nerve roots making up the plexus or the nerves which take origin from it, plus roentgenographic or other evidence of involvement in this region.

The sacral plexuses are formed by the lumbosacral trunk from the fourth and fifth lumbar nerves and the anterior divisions of the first, second, and third sacral nerves (Fig. 4). The sacral plexus lies, in 90 per cent. of cases, on the anterior surface of the piriformis muscle, and in 10 per cent. of cases it lies within the muscle. The sacral plexus lies behind the pelvic fascia and the branches of the hypogastric artery. It is also dorsal to coils of intestine, with the sigmoid portion of the colon in front of the left plexus and the lower part of the ileum in front of the right plexus.

When the fibers of the sacral plexus pass through the piriformis muscle and a spasm of this muscle occurs, according to Freiberg, sciatic pain may occur. The diagnosis is made by palpation of the spastic muscle by rectum and relief may be obtained by massage and physiotherapy to relax the spastic muscle, or by section of the muscle. Lesions of the sacral plexus may result from obstetrical injuries or injuries caused by forceps, bullet wounds, fractures of the head of the femur, or pelvic tumors. The diagnosis is made by recognition of involvement of the nerve roots which make up the plexus or its branches, or the nerves that take their origin from it, plus roentgenographic or other evidence of lesions in this region.

Lesions of the peripheral nerves may be caused by inflammatory affections of the nerves, trauma, pressure, or degenerative changes, and they may produce lesions which produce conditions commonly spoken of

under the heading of "neuritis". Inflammation of nerves may be primarily parenchymatous, or they may involve the sheath or the interstitial connective tissue. Thus, there is parenchymatous neuritis, perineuritis, and interstitial neuritis. However, a sharp difference cannot be established between these forms, since in all of them the parenchymatous nerve elements are involved to some degree. Interstitial neuritis or perineuritis usually is associated with true hyperaemia of the sheaths of the nerves, accompanied by transudation of serum and exudation of hematogenous leukocytes and lymphocytes. The nerve is red and swollen and the myelin sheath and the axis cylinders show some evidence of degeneration. This results in a typical degeneration of the peripheral portion of the nerve and considerable retrograde change of the central part, although the nerve sheaths are not affected.

Symptoms of neuritis vary, of course, with the severity of the affection. In perineuritis there is an extreme amount of sharp, lancinating, boring pain down the course of the nerve. Chills and fever with leukocytosis and general malaise may accompany the process. The pain is severely increased by movement of the part. The nerve trunk is swollen, frequently tender and palpable. The skin of the region supplied by the cutaneous nerve may be tender to pressure and show vasomotor changes. Sensory loss, motor weakness and paralysis, and areflexia occur.

A study of a group of patients suffering from true sciatic pain gives the impression that the term "neuralgia" should no longer be used to describe this type of pain, because most of such instances of pain probably are the result of neuritis of the sciatic nerve or its branches. Injuries to the peripheral nerves may produce the well-known phenomenon described by S. Weir Mitchell as "causalgia". This condition is most common in the hand following injury to the median nerve, but may occur in the foot or along the course of the sciatic nerve. Causalgia seldom appears when the nerve is cut completely. It occurs in the presence of incomplete injuries to the peripheral nerves when the physiological continuity of the nerve has been affected minimally. Objective sensation, although difficult to test for because of the extreme pain present, may appear to be intact. Hypalgesia, hypaesthesia, or hyperalgesia and hyperpathia may be encountered. It has been suggested that causalgia might be sympathetic in origin because of the associated vasomotor disturbances, and the emotional response of the individual to stimulation.

SUMMARY AND CONCLUSIONS

The interesting subject of sciatic pain in relationship to neuroanatomy and physiology could be pursued at great length. In this paper we have endeavored to present the modern concepts of the outstanding causative factors in the production of such pain. That new causes for sciatic pain will be discovered in the future and that further modification of our present ideas will become necessary is inevitable. At present, however, it is undoubtedly true that more patients who have this dis-

tressing symptom of sciatic pain can be relieved of their pain than was the case at any previous period of medical history.

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CONSERVATIVE TREATMENT OF SCIATIC PAIN IN LOW-BACK DISABILITY

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This paper is a description of the conservative treatment and a report of the results in 1000 patients seen for sciatic pain and low-back disability during the past ten years. By conservative treatment is meant the use of rest in special positions, support, medications, and physiotherapy. It does not include manipulative and operative procedures. In this group of 1000 patients there were 390 males and 610 females. There were fifty-two under twenty and eighty-two over sixty years of age. It is difficult to divide low-back pain into acute and chronic. In 519 the low-back pain had been present for two weeks or less, and in 481 it was of longer duration. The affections of 576 of these 1000 patients were diagnosed primarily as muscular or ligamentous strains in the region of the low-lumbar spine and sacrum. In 325 others a ligamentous strain was diagnosed in conjunction with disease or deformity of the vertebrae.

Sciatic pain, like other symptoms in low-back disability, was not a constant finding. At the time of examination, radiation of pain through branches of the sciatic nerve was present in 449 of these patients as follows: in 410 to the cutaneous distribution of the posterior femoral cutaneous and common peroneal nerves; in twenty-eight to the cutaneous distribution of the sural and superficial peroneal nerves; in seven to the distribution of the tibial nerve; and in four to the posterior femoral cutaneous distribution of both legs. In forty-three patients the radiation was along the middle and inferior gluteal nerves; in nine, the pain was complained of in the cutaneous distribution of the anterior femoral cutaneous nerve; and in four, the radiation was along the cutaneous distribution of the lateral femoral cutaneous nerve (Table II). In 409 of these patients the cutaneous radiation of pain could be interpreted only as a reflex phenomenon, a referring of pain from injured structures to the cutaneous distribution of the same nerve roots. Only approximate localization of the lesion from the referred pain was possible. In ninety patients there was extensive arthritic proliferation about the neural foramina with possible impingement upon the nerve roots. In six patients lesions within the spinal canal, which caused pressure upon the nerve roots, were demonstrated.

In most instances of low-back pain, the lesion causing the pain is presumed to be a ligamentous tear or a separation of fibers at the musculotendinous junction[5] (Fig. 1). As evidence for this assumption we have local tenderness over ligamentous structures. In a few instances there are superficial ecchymoses. Novocain injections into the most tender areas often produce temporary relief. Disappearance of the pain and disability is frequently observed following immobilization and support with attempted relaxation of the injured part of the back. Roentgenograms for

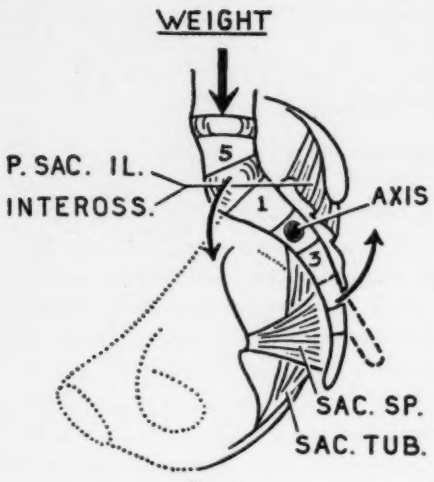

FIG. 1

Shows the common points of strain at the lumbosacral region and sacrum.
P. Sac. Il.: posterior sacro-iliac ligament;
Sac. Sp.: sacrospinous ligament;
Sac. Tub.: sacrotuberous ligament.
(From A Method of Anatomy, Descriptive and Deductive, by J. C. B. Grant, William Wood & Company, 1937. *Used by permission of Dr. J. C. B. Grant and The Williams & Wilkins Company.*)

soft-tissue detail are invariably negative. There are no early pathological studies recorded and operative procedures upon these patients are always performed too late to show any soft-part lesion.

A number of methods have been described recently for arriving at a more precise understanding of the pathological lesion responsible for the low-back pain. Steindler's studies aided in tracing the elusive referred pain from injured ligaments or the articular capsule. Operative attempts at muscular and ligamentous release have given suggestive evidence of the location of the lesion[7]. The work of Pitkin and Pheasant has pointed to the ligaments about the sacro-iliac and lumbosacral joints as the cause of the pain. In recent studies in a small number of patients the source of the pain has been found in the spinal canal[1] or in the neural foramina. Discussion of articular displacements or subluxations will not be undertaken, since the treatment of these does not come primarily under conservative therapy. Even the minor ones, described recently by Mennell, or those believed by the osteopath to be present in low-back pain, cannot be demonstrated clinically or roentgenographically.

It is recognized by anthropologists and by students of anatomy that the region of the spine at its attachment to the sacrum is frequently insecure and potentially weak [10]. Congenital anomalies are common at this area, and in addition there are many diseases, such as arthritis, which limit motion and lead to sprains and other injuries. Roentgenographic examination of these patients showed 375 abnormalities of the lumbar spine and sacrum in 323 of the 1000 patients. The anatomical and pathological changes are shown in Table I.

All of the congenital anomalies and many of the pathological changes, which interfered with the normal stability and balance of the spine, had been present for a long time before low-back pain appeared. In the presence of congenital anomalies, and faulty body mechanics which hasten the appearance of symptoms by causing malalignment of the spine and inefficient functioning of the supporting structures [3], a slight trauma, such as a sudden unguarded movement, may produce a severe sprain of the low back. The instability induced in the low back by the faulty body

TABLE I

ANATOMICAL AND PATHOLOGICAL CHANGES IN THE VERTEBRAE

Hypertrophic arthritis	96
Atrophic arthritis	70
Lumbosacral transverse joint	64
Spina bifida	50
Defective articular facets	45
Six lumbar vertebrae	14
Structural scoliosis	7
Fracture of lumbar spine	6
Metastatic malignancy	3
Epiphysitis of the spine	3
Spondylolisthesis	3
Tuberculosis of the hip	2
Tuberculosis of the spine	2
Paget's disease	2
Narrowing of the lumbosacral disc	2
Fused spine	2
Fracture of the pelvis	2
Short leg	1
Thirteen dorsal vertebrae	1
	375

mechanics is shown in roentgenograms by the tendency of the sacrum to assume a more nearly horizontal position, by the thrust of weight coming upon the articular facets at the limit of extension, by the increase in the lordosis with overriding spinous processes, and by the rotation and marked downward displacement of the lower ribs (Fig. 2).

What can be regarded as adequate treatment of the usual cause of low-back disability,—a muscular or ligamentous tear in the supporting structures of the lumbar spine and pelvis? It would be reasonable to believe that the treatment would be similar to that applied to muscular and ligamentous tears elsewhere in the body,—that is, rest with the injured structures in a position of relaxation until healing is at least partially completed. The author does not know how long it requires for a ligamentous tear to heal completely. From studies by Dehne [2] upon ligaments of dogs of a similar size, it should take at least two months.

What is the position of relaxation? Here we have considerable clinical and anatomical data. It is possible to secure relaxation of most of the posterior ligamentous structures about the sacrum and lumbar spine by supine recumbency with the lumbar spine flat, and with slight flexion (20 to 30 degrees) of the hips. This has been demonstrated in the cadaver. From observations upon the cadaver or the partially dissected lumbar spine and pelvis, the author can find no mechanical basis for traction upon the legs in extension for relief of the usual types of low-back pain. It does not aid ligamentous relaxation in the low back, or relieve muscular spasm in the lumbar region, but it does lead to immobilization of the spine in faulty positions.

The best method of securing immobilization and ligamentous

FIG. 2

Lateral roentgenogram of lumbar spine and pelvis, showing marked forward inclination of the pelvis with the sacrum approaching the horizontal. The articular facets are used at the extreme of extension. There are impinging spinous processes and depression of the lower ribs.

relaxation for the low back is to keep the patient constantly on a firm bed with the entire body horizontal except for slight flexion at the hip joint. This can be secured in the supine position by placing a pillow under the knees, or in the prone position, under the abdomen (Fig. 3). With very severe ligamentous injury it may be necessary to apply a plaster back shell or a plaster spica to hold the patient more continuously in this position temporarily. Only in the mildest of low-back injuries is the patient permitted to be ambulatory. The common tendency is to underestimate the severity of the injury.

In most instances while the patient is in bed, physiotherapy, consisting of heat daily, should be given to the posterior surface of the spine and pelvis to improve the local circulation, to relax muscle spasm, to relieve pain, and to hasten the absorption of inflammatory exudate [4]. This can be applied in a position of ligamentous relaxation with the patient prone by placing a pillow under the abdomen. In most instances it will be possible to permit the patient to sit up for meals and to use a cabinet chair for bowel evacuation without disturbing the healing process. As soon as muscle spasm and pain begin to subside and the patient can move in the bed without much discomfort, exercises can be given to the patient to teach him muscular coordination so that he may improve the balance and carriage of the entire body to prevent further strain. The period that the patient must be kept recumbent varies greatly. It is usually longer in chronic conditions than in acute ones. The average period of complete recumbency in this group was ten days. Complete recumbency should be continued until pain and local tenderness have almost entirely subsided.

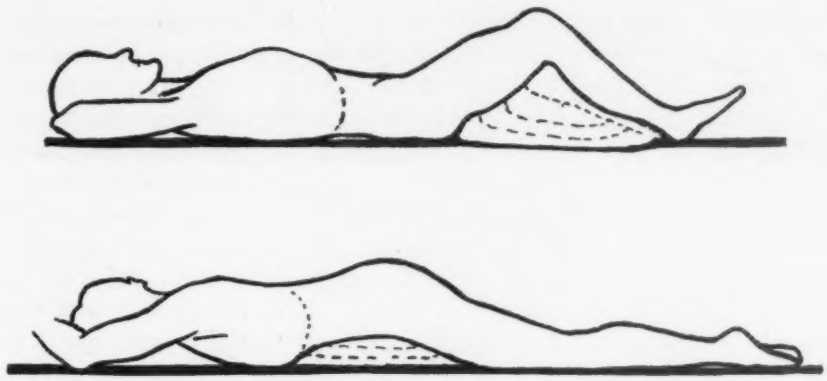

FIG. 3

The positions assumed by the patient in bed to relax the muscles and ligamentous structures of the low back and the sacrum.

This does not mean the end of treatment. Usually a prolonged period of convalescence is required until the individual is again able to assume his usual activities. Physiotherapy in the form of heat, exercises, and recumbent rest in positions which do not sprain the low back for part of the day are continued for a longer period. If the patient is not pushed beyond his physical capacity and if the ligaments are prevented from being overstretched during healing, a gradual improvement in symptoms, in muscular tonus, and in good carriage should be observed.

Because it requires a prolonged period to heal a ligamentous tear and because recurrence of the lesion may occur by an unguarded movement during convalescence, a spinal support is usually required. There are many kinds which can be effective if they are applied properly. The following are the criteria of a good spinal support:

1. It should be long enough to extend several vertebrae beyond those attached to the sprained ligaments.
2. It should be firmly attached to the pelvis.
3. It should be fitted to hold the joints of the lumbar spine in a position midway between flexion and extension, the optimum position for function (Fig. 4).

Treatment should attempt to relieve the immediate disability and also should do whatever is possible to avoid a recurrence. This will include improvement of general health, reduction of obesity, better habits of work and leisure, the easing of social maladjustments, and many others. In regard to the spine itself, prolonged guidance as the patient is beginning to resume his work is important. The spinal support should be discarded gradually as the patient loses his pain and limitation of motion, and learns how to use the body habitually in a position which is not at the extreme of extension, but midway between full extension and flexion. This is the position seen in good body mechanics.

With such treatment continued until subjective symptoms and objective signs subside what can be expected? In this group it required an

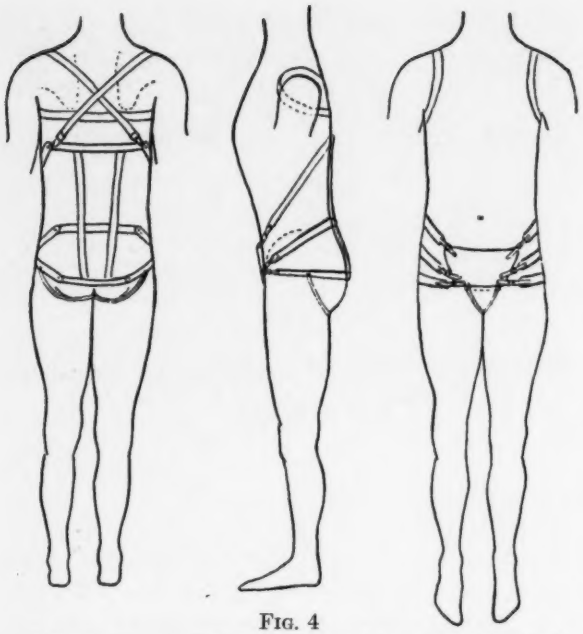

FIG. 4

Spinal support used during convalescence in the treatment of low-back pain. The shoulder straps are not used if there are no symptoms in the upper dorsal or cervical regions. Note the firm grasp of the pelvis, the fitting of the support to hold the spinal joints in a position not at the extremes of their motion, and the avoidance of compression upon the supporting muscles of the back and abdomen. (From Body Mechanics in the Study and Treatment of Disease, by Joel E. Goldthwait, Lloyd T. Brown, Loring T. Swaim, and John G. Kuhns. Ed. 2 Philadelphia, J. B. Lippincott Co., 1937. *Courtesy of J. B. Lippincott Company.*)

average of forty-eight days before the spinal symptoms and signs subsided and the patients returned to their work, with extremes of one week and ten months. A large number of patients who return and continue at work are never wholly free of symptoms, although physical signs do not suggest any serious lesion in the spine. The reasons for their continued symptoms are varied. It may be a chronic arthritis, or a congenital anomaly making a potentially weak back, or working long hours with the back bent, or the inability of the patient to learn to use the body in a more correct habitual position. A permanent muscle weakness may lead to slight recurrent strain when the patient works too strenuously. Certain of these recurrences can be relieved by more radical procedures, but often these slight symptoms will recur even after surgical correction of the supposed cause.

The effect of conservative treatment in the relief of both local and referred pain is shown in Table II. Relief of pain was observed in 771 of the entire group. In those with sciatic radiation of pain, the pain

TABLE II

RESULTS OF CONSERVATIVE TREATMENT IN THE RELIEF OF PAIN

	No.	Relieved	Unrelieved	Unknown
Local pain only	495	374	35	86
Sciatic radiation	449	356	31	62
Gluteal radiation	43	31	5	7
Anterior thigh radiation	9	6	1	2
Lateral thigh radiation	4	4	0	0
Totals	1000	771	72	157

disappeared in 356 of 449 patients, or about 79 per cent. There was no appreciable difference in the relief of pain in the various cutaneous distributions. The alleviation of pain is not the only factor which determines whether the individual is disabled from lesions in the low back. If the pain is not severe the patient will often continue at his usual activities.

If this study is to be of value it should suggest which types of low-back pain will probably be relieved under conservative treatment (Table III) and which will probably not be helped by conservative treatment alone. The types of low-back disability can be placed in four large categories:

1. *Acute Traumatic and Chronic Postural Strains of the Low Back.* All of these patients can be expected to recover under conservative treatment. The length of time that treatment must be continued will depend upon the severity of the trauma, the chronicity of lesion, and the cooperation of the patient.

In a small number of patients a change from full extension of the spinal joints to one of relative flexion cannot be obtained by conservative measures alone. This can be demonstrated by lateral roentgenograms (Figs. 5-A and 5-B), with the thighs extended and flexed upon the abdomen. If, after two weeks of adequate, conservative treatment, no motion can be demonstrated in the spinal joints of the low back, other measures, most commonly manipulation of the low back, are indicated. After normal mobility has again been obtained in the low spinal joints, conservative measures should be resumed until the patient has recovered.

2. *Structural Changes in the Vertebrae or Their Contiguous Structures.* If the normal osseous contour is again established and adequate fixation is given, conservative treatment is needed to recover normal function. In other diseases where there is much deformity or disturbance of stability, an attempt should be made to correct this; then conservative treatment may be given. Where structural restoration cannot be expected, as in metastatic malignancies, conservative therapy may prolong function and is of great help in palliative treatment.

3. *Low-Back Pain from Pressure or Irritation within or about the Spinal Canal.* In this group conservative treatment may be of little avail. Here, if there is a definite lesion—namely, a ruptured nucleus pulposus or a spinal cord tumor—its removal is indicated.

TABLE III

RESULTS OF CONSERVATIVE TREATMENT IN THE RELIEF OF DISABILITY

	No.	Improved	Unimproved	Unknown
Acute traumatic and chronic postural strains	904	744	47	112
Structural changes in the vertebrae	90	47	8	35
Pain from pressure or irritation within the spinal canal	6	0	6	0
Pain referred from other parts of the body	0	0	0	0
Totals	1000	791	61	147

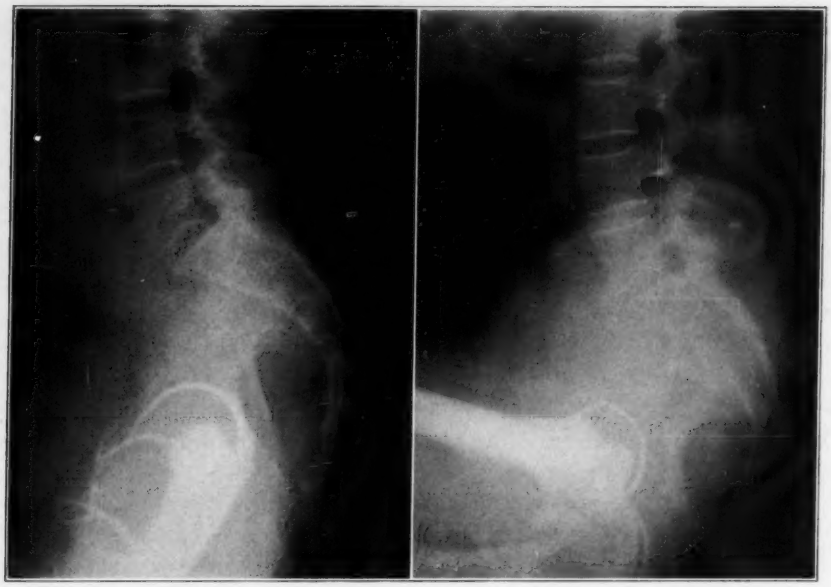

FIG. 5-A FIG. 5-B

Lateral roentgenograms of the lumbar spine and pelvis.

Fig. 5-A shows the position of the sacrum and articular facets with the thighs extended.

Fig. 5-B shows good mobility in the lower spinal joints when the thighs are flexed upon the abdomen.

4. *Pain Referred to the Back from other Parts of the Body, Usually the Abdominal and Pelvic Viscera.* There were no patients in this 1000 cases with low-back pain of this type. Here the lesion, which causes pain referred to the low back, should be treated.

SUMMARY

Low-back disability with sciatic radiation may arise from an infinite number of causes. The most frequent cause is injury to the ligaments, muscles, and fascia supporting or investing the low back or pelvis. The cutaneous radiation is most commonly a reflex phenomenon, a referring of pain from deep structures to the cutaneous distribution of the same nerve root. The lumbosacral region of the spine is the weakest portion of the vertebral column. Contributing factors to sprains at this area are anomalies and diseases of the vertebrae and faulty habits of use of the body. Faulty body mechanics leads to impaired functioning of the muscle supports, defects in balance against gravity, and the tendency of the low spinal joints to be used at the limit of their extension.

Adequate conservative treatment should include rest of the entire spine in a good mechanical position on a firm surface, heat applied to the injured portion of the spine, and, when movements can be performed with relative comfort, exercises to teach the patient how to use the body habitually in good body mechanics. Later when the patient is first ambula-

tory, a support should be fitted with the patient's body in the best corrected posture possible, so that strain will not come upon the healing structures.

Pain referred along the sciatic nerve is relieved by such treatment in approximately 79 per cent. of all patients who present this symptom. When we group patients according to the cause of the disability we find that low-back disability caused by ligamentous and muscular strains responds most frequently to conservative treatment.

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POSTERIOR PROTRUSION OF THE LUMBAR INTERVERTEBRAL DISCS *

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For many years orthopaedic surgeons have been studying the problem of low-back and sciatic pain, and have recognized that in the majority of cases the cause of the patient's disability is mechanical. An exact classification of the various types of mechanical derangement is impossible unless the pathological anatomy can be demonstrated either on the operating table or at autopsy. Within the past decade it has been discovered that one of the common mechanical derangements causing sciatica is posterior protrusion of the lumbar intervertebral discs. The increase in the number of patients operated upon for this condition is shown in Table I.

PATHOLOGY

The limited time at the authors' disposal precludes a complete discussion of the anatomy and pathology of intervertebral-disc lesions. The recent reviews by Saunders and Inman are both comprehensive and accurate. The normal intervertebral disc cushions the central nervous system from trauma and serves to transmit the body weight from one vertebra to another and to permit mobility of the spine. Each disc possesses a certain turgor or internal hydrostatic pressure, which, as measured by Petter, amounts to thirty pounds for the lumbar discs. This internal pressure is, of course, markedly increased during certain of our normal activities; for instance, if a man is lifting a 100-pound weight, the lumbar intervertebral discs are subjected to the weight of the body above them, to the 100-pound weight, and, in addition, to the compressive force exerted by the trunk muscles contracting between the pelvis and the thoracic cage. Extrusion of the nucleus pulposus may occur in any direction following weakening of its surrounding tissues by developmental defect, disease, degenerative change, or trauma. If the cartilaginous end plate is weakened, protrusion may occur into a vertebral body. Although such *Knorpelknötchen* may cause local backache, they apparently never cause radiating pain.

* The statistical data of this paper are compiled from the records of 139 consecutive proved cases of posterior intervertebral-disc protrusions in the lumbar region, operated upon at the Massachusetts General Hospital for a chief complaint of "sciatic" pain. A few cases reported in previous papers have been omitted, either because they were operated upon at other hospitals and end-result data are not available, or because on restudy they did not seem to fit the syndrome. This accounts for various minor statistical discrepancies between this and others of our papers.

TABLE I

NUMBER OF OPERATIONS PERFORMED FOR POSTERIOR PROTRUSION OF LUMBAR INTERVERTEBRAL DISCS AT THE MASSACHUSETTS GENERAL HOSPITAL UP TO JANUARY 1940

Year	No.	Year	No.
1925	1*	1934	5
1927	1*	1935	8
1930	2*	1936	23
1931	1*	1937	19
1932	5	1938	27
1933	1	1939	48†
		Total	141†

* These were recognized as ruptured discs only after restudy of the histological material in 1932.

† Includes two recurrences.

Posterior protrusions in the low lumbar region sufficiently large to press on one or more roots of the lumbosacral plexus cause a very definite train of symptoms and signs, which have been described in detail in previous communications. In a series of 155 cases of proved intervertebral-disc protrusion into the spinal canal, verified by operation at the Massachusetts General Hospital, 139, or 90 per cent., occurred in the lumbar region, the fourth and fifth lumbar discs being the favored locations. The lesion is laterally placed, usually directly beneath the articular facet, and, when of sufficient size compresses the nerve root between the ruptured disc and the facet just before its exit through the intervertebral foramen. A lesion at the fourth lumbar disc invariably compresses the fifth lumbar root, while a lesion at the fifth lumbar disc invariably compresses the first sacral root, providing the protrusions are of sufficient size to cause any nerve-root pressure. This is due to the fixation of the root in its dural sheath at its point of exit through the intervertebral foramen. The other roots lying free in the cauda equina are easily displaced to one side and so avoid pressure in most instances. Occasionally the lesion is so large that it completely occludes the dural sac. The lesion, as seen at operation, usually presents itself as a small, elastic, pea-sized nodule lying beneath the posterior longitudinal ligament and on incision of the ligament can be removed as a rolled-up, completely detached bit of intervertebral-disc tissue. Occasionally, the fragment is still attached to the disc but produces a localized protrusion. Rarely there is a generalized backward bulging or prominence of the whole posterior disc margin with a resultant transverse indentation of the ventral surface of the dural sac. The nerve root impinged upon is, of course, definitely flattened at the point of pressure, and there is oedema and injection of the involved nerve root proximal to the level of the lesion. There is associated local thickening of the ligamentum flavum in a fairly high percentage of cases of proved ruptured discs. Abnormal mobility of the involved interspace may be demon-

strated in some of the cases, and in a few instances, on opening the dura, chronic adhesive arachnoiditis has been demonstrated.

ETIOLOGY

There seem to be two possible explanations for the presence of these protrusions. They may be due to sudden rupture of the annulus fibrosus, the result of raising the internal disc pressure above the breaking point of the annulus fibrosus, with protrusion of the nucleus pulposus. The history of certain of the authors' patients would lead to the belief that this can and does occur clinically. It is analogous to the blow-out of an automobile tire.

A second explanation is that a fissure occurs in the annulus fibrosus as the result of the wear and tear of ordinary use or of degenerative change, and through this fissure there is a slow, gradual extrusion of disc tissue with final resultant symptoms of sciatica when the extruded mass becomes large enough to press upon a nerve root. Clinical cases of this type may have recurring episodes of low backache over many years before the extruded mass becomes large enough to precipitate an attack of sciatica.

Finally, there are small posterior protrusions which Schmorl found in about 15 per cent. of autopsies, and which he considered to be congenital in origin and of no clinical significance. There can be no doubt of the existence of such congenital lesions, but they seem to bear no direct relationship to the ruptures described here, as they occur in a higher percentage of females than of males and show no predilection for the low lumbar spine.

The symptoms and signs of a ruptured lumbar intervertebral disc are most variable, but the general pattern is so uniform that the presumptive diagnosis can often be made without contrast myelography. A common type of case history follows:

A vigorous man in his thirties was perfectly well until, while lifting a heavy weight, he felt something "snap" in his low back and had immediate pain in the lumbosacral region. Sometime thereafter he developed "sciatica" in one leg, which he describes as a deep-seated pain beginning in the buttock, radiating down the posterior thigh, the posterolateral aspect of the calf, and occasionally going into the lateral border of the foot or the great toe. Coughing and sneezing, bending forward to tie the shoes, and lying face down in bed cause a marked increase in the radiating pain. If an uncomfortable position is maintained for a time, the leg tingles, and the foot may "go to sleep". There have been two or three separate periods of disability, the first ones relieved by rest in bed; the last one, however, has not yielded to conservative measures including back-strapping, heat, bed-rest, plaster casts, corset, and osteopathic manipulation. On examination the patient is found to stand with his trunk thrust forward and to one side, with most of his weight borne on the non-painful leg—"sciatic scoliosis". The normal lumbar lordosis has been lost, and in its place is a fixed reversal of the lumbar curve, with prominence of the spinous processes of the third, fourth, and fifth lumbar vertebrae. All motions of the lumbar spine are considerably restricted by involuntary muscle spasm. The patient's finger tips cannot possibly be made to touch the floor if the knees are kept straight. The fixed list of the spine and pelvic tilt are uninfluenced by a lift under either foot. Forced hyperextension causes severe radiating pain. In the sitting position, the trunk can be flexed at the hips with ease, so that the chest approaches the knees, but

observation reveals that the lumbar spine remains in its fixed position and goes forward *en bloc*. The list of the lumbar spine may not be so noticeable, but side bending remains restricted.

The patient gets into bed or on to the examining table with extreme care. Turning over on the table is an ordeal, and he is unable to lie face down until a fat pillow is placed beneath the abdomen. In that position on palpation there is found definite local tenderness in the mid-line between the fourth and fifth lumbar spinous processes or at the lumbosacral junction. Percussion over these spinous processes may cause radiating sciatica, as may jugular compression (Naffziger's sign). There may be a little tenderness at the sacrosciatic notch on the affected side and over the course of the sciatic nerve. Inspection reveals some atrophy of the buttock, thigh, and calf on the painful side. The ankle jerk is usually diminished or absent on the painful side, and normal on the other. Peripheral, sensory, or motor changes are found in less than half the cases. Straight-leg raising may be somewhat limited on the non-painful side, and is markedly limited on the painful side. Lasègue's sign may be positive.

A history and physical examination similar to this suggest strongly that the diagnosis is posterior protrusion of one of the lower lumbar intervertebral discs.

The clinical differentiation of lesions at the lumbosacral junction from those one or two discs higher is extremely difficult, and no really reliable method is yet available. The distribution of the radiating pain does not correspond to the sensory dermatomes. Spurling and Bradford suggest that if subjective or objective sensory changes are present (that is, paraesthesia, hypaesthesia, or anaesthesia) in the anterolateral calf and great toe, the lesion is between the fourth and fifth lumbar vertebrae, while involvement of the lateral calf and lateral border of the foot indicates a lumbosacral lesion. Although this differential diagnosis would be most useful, the authors in their series of cases have not been able to verify its reliability. Their statement that the ankle jerk is absent or diminished if the lumbosacral disc is involved, and present if the disc between the fourth and fifth lumbar vertebrae is involved is also not a reliable diagnostic differential point.

The usual anteroposterior and lateral roentgenograms are of some diagnostic aid. They may be entirely normal, as the displaced disc tissue casts no roentgenographic shadow, and may be too small in volume to cause apparent narrowing of the intervertebral spaces. However, a definitely narrowed space between the fourth and fifth lumbar vertebrae is of some diagnostic importance. A narrow lumbosacral interspace occurs about as frequently as other congenital abnormalities, such as sacralized transverse processes and spina bifida, and should be considered as an incidental finding unless there are other changes present, such as osteosclerosis or spur formation, to suggest that the narrow disc is not congenital but acquired. If the fifth lumbar vertebra is sacralized, the rupture is almost invariably above it. The scoliosis and kyphosis noted clinically are also seen on the roentgenogram.

The next step in diagnosis is the lumbar puncture, which should be done as low as possible, preferably at the lumbosacral interspace. The first two cubic centimeters of fluid removed should be sent as a separate

specimen for determination of the total protein. After careful manometric tests to rule out dynamic block, further fluid is withdrawn for cytological study, and for Wassermann and colloidal-gold tests. A total protein higher than 35 or 40 milligrams per 100 cubic centimeters is confirmatory evidence that the pathological process is intraspinal. The authors have a few proved cases in their series which have had normal total protein, but the average figure has been above 60 milligrams per 100 cubic centimeters. As the lesion is usually at one of the two lowest intervertebral discs, the lumbar-puncture needle is seldom placed caudal to it, and it is rare to find any dynamic block.

The differential diagnosis between protruded intervertebral discs and other causes of sciatica cannot be discussed here. It must not be forgotten that sciatic pain of any origin is accompanied by limitation of motion of the lumbar spine, limitation of straight-leg raising, and diminution or absence of the ankle jerk. There is no single symptom or sign from which the diagnosis can be made. There is no easy road to correct diagnosis which can supplant the time-honored method of careful history-taking, complete physical examination, and the evaluation of that information in the light of appropriate laboratory tests. Malignant tumors, primary and metastatic, infectious lesions of the low back, including the sacro-iliac joints, arthritis, including the Strümpell-Marie as well as the hypertrophic types, must all be considered among the organic causes. On the other side of the ledger are all the strains, sprains, and subluxations of the lumbosacral and sacro-iliac joints, and of the ligaments and muscles controlling them. Diagnostic problems and errors must arise to plague all of us, even when the combined skill of the internist, neurologist, roentgenologist, and orthopaedist are directed toward the solution of the problem.

Preoperative diagnosis and localization of the lesion by means of contrast myelography is the next procedure to be considered. Iodized oil (lipiodol) is considered by competent roentgenologists [4, 6] to be the most satisfactory medium. Its use led to accurate preoperative localization in most of our cases, and without it the evolution of our knowledge of this clinical entity would have been much retarded. However, in five cases the lipiodol roentgenographic examination was positive, but the exploratory operation proved to be negative. In two of the nine cases in which the lipiodol roentgenographic examination was negative, ruptured discs were found at the time of the exploratory operation.

There has been a good deal of acrimonious discussion concerning the use of a contrast medium such as lipiodol for the localization of intraspinal lesions. Spurling suggests that clinical diagnosis and localization may be so accurate that the use of a contrast medium is unnecessary. Chamberlain and his coworkers state that lipiodol is dangerous and that air or oxygen myelography yield exact diagnostic information.

In a series of cases in which the authors have used both air and lipiodol myelography, they have found that the former is diagnostically accurate in not over 50 to 60 per cent. of the cases, while lipiodol is

accurate in over 90 per cent. The authors sometimes make air or oxygen myelograms at the time of the lumbar puncture, and if unequivocal, positive information is obtained, they proceed with the operation, but if the myelograms are negative or doubtful, lipiodol is injected a few days later. The lipiodol examination gives so much more accurate, dependable information that many workers have stopped using air or oxygen myelography. Lipiodol in the authors' hands and in the experience of other competent observers has proved to be the best of contrast media so far available. The risk of complications from its use is not great, and if it is reserved for those cases in which conservative treatment has failed, it becomes a preoperative localizing diagnostic test reserved for patients in whom clinical and other laboratory examinations make the diagnosis of a lesion in the low-lumbar spine almost certain. Under these circumstances, a lesion will be demonstrated and accurately localized in a very high percentage of cases. In two cases of this series no lesion was demonstrated on lipiodol examination, but exploratory laminectomy revealed a ruptured lumbosacral disc pressing on the first sacral nerve root, but not encroaching on the thecal sac. The lipiodol is always removed as completely as possible at the time of the operation. The technique of lipiodol and pneumomyelography has been described elsewhere. Thorotrast seems to the authors to entail too much risk to warrant its use. They have hesitated to recommend exploratory laminectomy unless the lesion has been diagnosed and localized by myelography. Occasionally such a procedure is justified and may become more so as greater skill in clinical diagnosis and localization is acquired. The discovery of an absorbable, innocuous, contrast medium of the density of lipiodol is awaited.*

METHODS OF TREATMENT

The authors' cases are selected for operation on the following basis: Operation is reserved for those who have had adequate conservative treatment under competent direction without satisfactory relief of symptoms, and for those who, although they may have had no conservative treatment, have definite evidence of serious nerve-root pressure (areas of anaesthesia, motor weakness, or paralysis, loss of sphincter control, etc.). The operative technique has been described by Mixter, Love, Fincher, and others. Suffice it to say that a partial laminectomy, in which adjacent portions of two laminae are removed but the continuity of no whole lamina is sacrificed, is usually sufficient for exposure and removal of the lesion and removal of the lipiodol if it has been left in the lumbar sac. If marked abnormal mobility is demonstrated at the site of the lesion, or if an articular facet has been sacrificed, the involved area (the fourth lumbar to the first sacral vertebrae) is fused. This can be easily performed on the side opposite the lesion. A modified Hibbs method reinforcing the fusion

* Since this paper was read, a satisfactory method for the removal of lipiodol has been worked out[7]. This makes it unnecessary to operate on those cases that have had negative lipiodol examination and obviates the danger of late arachnoiditis due to irritation from retained lipiodol.

with an osteoperiosteal graft and additional bone chips from the tibia is used. No attempt is made to bridge the laminectomy defect. There is a definite place for fusion in selected cases, and about one-third of this series of cases are fused. Whether fusion is performed or not, strenuous activity, heavy work, etc., are not permitted for six months after operation. It is unnecessary to emphasize that muscular rehabilitation by means of carefully graded exercises should be an integral part of the post-operative care of every patient. Details of after-care vary with the physician in charge.

CONSERVATIVE TREATMENT

There is a good deal of clinical evidence leading to the conclusion that the lesion under discussion is by no means rare and that many patients with low-back pain and sciatica, whose symptoms are mild or who recover under conservative treatment, have a protruded disc as the cause of their symptoms. The only way of obtaining exact evidence on that would be to inject lipiodol into the spinal canal of a large consecutive series of cases of low-back strain and to follow them carefully without operation. Ten patients in our series with a typical or suggestive defect, as shown by examination with lipiodol, and a large number of others who clinically seemed to have protruded discs have been treated conservatively. The use of a plaster jacket applied with the lumbar spine in slight flexion seems to be the most efficacious method of immobilization. The patient whose symptoms are not relieved within a few weeks by bed-rest or by the application of such a jacket usually requires surgery. As has been stressed before, this series of proved cases have had every form of conservative and operative treatment tried, including belts, braces, jackets, corsets, fasciotomy, sacro-iliac fusion, etc. The point that is not known is how many additional cases of protruded disc were saved from surgery by such treatment. Manipulation of the spine in suspected disc cases is strongly condemned. It may result in paraplegia.

ANALYSIS OF CASES

Age and Sex. The age of the patients averaged thirty-seven years and ranged from sixteen to sixty-five. Seventy-eight per cent. were males and 22 per cent. females.

History. In 50 per cent. disability occurred immediately following injury and in another 30 per cent. the disability developed after a latent period. In the order of frequency, the most common causes of injury were lifting a heavy weight, falling from a height, and incurring a twisting strain.

Pain. In 60 per cent. the pain was constant from onset until operation, in 40 per cent. there were remissions and relapses. In 30 to 40 per cent. backache preceded sciatica. In 80 per cent. the referred pain was unilateral, and in 20 per cent. bilateral. Ninety-five to 100 per cent. of the patients complained of pain in the posterior and lateral thigh; 90 per

cent., in the posterior and lateral calf; 70 per cent., in the lumbosacral region; 65 per cent., in the gluteal region; and 20 per cent., in the lateral border, sole, or dorsum of the foot, or the great toe.

Physical Examination. Straight-leg raising was limited on the painful side in 95 to 100 per cent., and on the other side in 30 to 50 per cent. Lasègue's sign was usually positive. Sciatic scoliosis was present in 60 per cent. (40 per cent. contralateral and 20 per cent. homolateral). Reversal of the usual lumbar curve (kyphos or flat back) and limitation of motion of the lumbar spine occurred in 90 per cent. There was local tenderness over the lumbar spinous processes at the level of the lesion in 50 to 60 per cent.; radiating pain on coughing, sneezing, percussion of the lumbar spinous processes, or jugular compression in 40 per cent.; and atrophy of the buttock, thigh, or calf in 50 per cent.

Neurological Signs. The ankle jerk was absent or diminished in 70 per cent. Sensory changes in the extremity—most commonly on the lateral calf, lateral border of the foot and fifth toe, and posterior thigh—occurred in 35 per cent. Motor weakness of the dorsiflexors of the foot or the gastrocnemius muscle was present in 15 per cent., and sphincter disturbance in 5 per cent.

Loçation of the Lesion. The lesion occurred at the third lumbar in two cases; the fourth lumbar in eighty-one; the fifth lumbar in fifty-seven; and the first sacral in one. These figures include two patients who had a recurrence of symptoms and a second operation.

Other Pathology. In twenty-three patients who were operated upon after a preoperative diagnosis of possible ruptured disc, hypertrophy of the ligamentum flavum was found in ten; chronic adhesive arachnoiditis, in eight; hypertrophied ligamentum flavum plus arachnoiditis, in two; hypertrophied ligamentum flavum plus generalized posterior bulge of the disc, in one; and varix over nerve root, in two.

END RESULTS

The two deaths in this series occurred in patients who had preoperative paraplegia from extremely large ruptured discs. Their symptoms did not respond to relief of the pressure. One of them died postoperatively of complicating sepsis. The other one lived five months after operation. Since the death of that patient in 1931, there have been no operative deaths in a series of over 130 consecutive cases.

Of 139 patients with protruded disc in the lumbar spine operated upon at the Massachusetts General Hospital up to January, 1940, ninety-four have been followed for at least one year, and the postoperative result ascertained. The average follow-up period was three and one-half years. Seventy-seven per cent. of the patients have had complete relief from their radiating pain, and an additional 18 per cent. have had only minor discomfort in the affected leg; in other words, the relief from the sciatic pain has been satisfactory in 95 per cent. of the cases. Five per cent. must be classed as failures. In this group are two patients who had

temporary relief from the removal of a ruptured disc. One had a recurrence of symptoms and at a subsequent operation was found to have a recurrent protrusion at the same site. The other patient developed sciatica in the opposite leg, and at a second operation was found to have a ruptured disc at the same level but on the opposite side from the original rupture. Both of these patients have been relieved of their symptoms by the second operations. Thirty-seven patients have been operated upon too recently for end results to be available, and six were untraced.

Back Symptoms. Although laminectomy and removal of the protruding disc fragment is almost uniformly successful in relieving the patient of his major symptom, radiating sciatic pain, there is another factor which has particular interest and which has been studied most carefully, and that is weakness or discomfort in the low back. The authors have found that 60 per cent. of their patients deny having any back symptoms, and consider themselves as strong physically as before the onset of their disability. Thirty-two per cent. complain of slight to moderate back weakness or discomfort, and 8 per cent. consider themselves markedly disabled. In other words, 40 per cent. would probably have had to seek lighter work if their occupation entailed manual labor. As the majority of cases of ruptured discs occur among vigorous young and middle-aged men, even slight to moderate back disability is serious. In the case of the laboring man, it may mean changing from self-support to economic insufficiency, while in others it means curtailing all vigorous sports. There are two probable explanations for the occurrence of a weak, painful back. The mechanical derangement due to the ruptured disc is obvious in certain instances. Roentgenograms may show marked narrowing of the vertebral interspace with accompanying hypertrophic spur formation, and subluxation of the articular facets; and so, although the protruded disc tissue has been removed and the sciatica relieved, there remains a *locus minoris resistentiae* easily susceptible to strain. Secondly, the laminectomy itself may weaken the back to a greater or lesser degree, particularly if sacrifice of an articular facet is necessary. In about one

TABLE II

RELIEF OF SCIATIC PAIN FOLLOWING OPERATION IN NINETY-FOUR PATIENTS FOLLOWED FOR ONE YEAR OR MORE

Results	Whole Series		Spines Fused		Spines Not Fused	
	No.	Per Cent.	No.	Per Cent.	No.	Per Cent.
Complete relief	72	77	30	91	42	69
Minor symptoms	17	18	2	6	15	25
Moderate pain	2	2	0	0	2	3
Severe pain	3	3	1	3	2	3
Totals	94	100	33	100	61	100

TABLE III

RESIDUAL BACK SYMPTOMS FOLLOWING OPERATION IN NINETY-FOUR PATIENTS FOLLOWED FOR ONE YEAR OR MORE

Results	Whole Series		Spines Fused		Spines Not Fused	
	No.	Per Cent.	No.	Per Cent.	No.	Per Cent.
Normal back	56	60	24	73	32	52
Mild weakness or pain	30	32	7	21	23	38
Moderate weakness or pain	5	5	1	3	4	7
Severe weakness or pain (pseudarthrosis)	3	3	1	3	2	3
Totals	94	100	33	100	61	100

out of three cases, the spine has, therefore, been fused. The criteria for fusion have varied with the personal convictions of the surgeon in charge, but in general, if the patient was a laboring man or indulged in active athletics; if, at operation, there seemed to be abnormal mobility of the involved vertebrae; and if the laminectomy was extensive or required sacrifice of an articular facet, fusion was performed. The results in the fused and unfused cases are compared in Tables II and III. Seventy-three per cent. of the patients having fusion considered their backs of normal strength, whereas only 52 per cent. of those without fusion had no complaints. The two patients who had recurrence of the disc protrusions, proved by operation (referred to above), had not had spine fusions done at the time of the first operation, and it is possible that an unfused spine is more likely to have further protrusion of disc tissue than one which is fused.

Ten cases of probable ruptured intervertebral disc with positive lipiodol examination have been treated conservatively. One is free of symptoms; the others have back and leg pain of greater or lesser severity.

There are certain drawbacks to spine fusion. It prolongs the operative time somewhat, subjects the patient to a certain additional operative risk and to a somewhat more prolonged convalescence. This series of cases is as yet too small to allow definite conclusions to be drawn, but there is a place for spine fusion at the time of laminectomy in perhaps 20 to 30 per cent. of the cases of protruded intervertebral discs.

The end-result study of twenty proved cases, in which there was compensation involved and the patients' medical expenses were paid by an insurance company, reveals that nine, or 45 per cent., have resumed their original occupation and consider themselves fully recovered. Six, or 30 per cent., are at work at lighter jobs. Five, or 25 per cent., are still receiving compensation or have litigation pending and consider themselves

disabled. Three additional patients have had recent operations, and the result cannot yet be determined. The fact that 75 per cent. of these cases have returned to some type of work leads to the belief that there is justification in expecting cooperation from insurance companies in the attempt to rehabilitate otherwise totally incapacitated patients.

There is a serious dilemma facing the medical profession with regard to who shall treat these cases, and although the situation has been discussed verbally, little has been put into writing. The neurosurgeon and the orthopaedic surgeon each have an interest in the situation. The neurosurgeon by reason of training, experience, and skill in handling nerve tissue is in general better equipped to perform the laminectomy than the average orthopaedic surgeon. However, he has had little or no experience in the recognition and conservative treatment of mechanical low-back disabilities, in the indications for and technique of spine fusion, and in supervision of the muscular rehabilitation exercise program which is so necessary in the postoperative care of the patients. The orthopaedic surgeon, by virtue of his training, has these qualifications. Who then shall care for cases of protruded intervertebral disc? It would seem that a competent orthopaedic surgeon should be placed in charge of every case of chronic mechanical low-back disability with or without sciatica. If conservative treatment fails or seems to him inadvisable, he should then call in consultation a neurosurgeon before lipiodol roentgenography or other complicated investigative procedures are initiated. The decision as to whether myelography is indicated, whether spine fusion should be done at the time of laminectomy, and so on, is discussed after the neurosurgeon has made a thorough examination of the patient. If spine fusion is indicated, the orthopaedic surgeon should join the operative team and do this part of the operation. He should also supervise the program of postoperative exercises designed to restore the patient's physique to normal.

SUMMARY AND CONCLUSIONS

Posterior protrusion of one of the lumbar intervertebral discs into the spinal canal is one of the most common mechanical derangements of the low back in patients suffering from intractable sciatic pain. The leg pain is due to direct pressure of the displaced intervertebral-disc tissue on one or more roots of the cauda equina. In addition to this lesion, there may be found associated thickening of the ligamentum flavum, chronic adhesive arachnoiditis, hypermobility of the involved vertebrae, and oedema of the involved nerve roots. Although the etiology of posterior disc protrusions is not perfectly clear, trauma to and degenerative changes in the intervertebral discs alone or in combination seem to be the usual causes of posterior protrusion.

Among the most common and characteristic symptoms and signs, are intractable sciatic pain following a lifting injury, accompanied by limitation of back motion and of straight-leg raising, sciatic scoliosis, and lumbar kyphosis, tenderness over the lower lumbar spinous processes, and

absence of the ankle jerk. The total protein of the spinal fluid is usually elevated. There are no changes on the routine roentgenograms characteristic of ruptured intervertebral discs, but narrowing of the fourth lumbar disc is of some importance if the clinical picture is characteristic. Lipiodol examination is highly accurate in making the correct diagnosis and in localization of the lesion, but because of potential danger, the use of lipiodol should be reserved for cases in which surgery is necessary. Pneumomyelography and clinical localization of the lesion may make the use of lipiodol necessary in not more than one-half the cases. Conservative treatment should be tried in every case of suspected protrusion of the intervertebral discs unless there is obvious serious nerve-root pressure, as shown by objective sensory or motor disturbance. Bed rest and immobilization of the lumbar spine in a plaster jacket seem to be the most effective means of conservative treatment.

Surgical treatment consists in the removal of the ruptured disc fragment through as small a laminectomy incision as possible. Spine fusion at the time of the laminectomy seems to give definitely better results than laminectomy alone.

Of ninety-four cases of proved ruptured intervertebral disc followed for at least one year after operation, 77 per cent. had complete relief of sciatic pain, and an additional 18 per cent. had only minor leg pain. There were two proved cases of recurrent ruptures in this series.

The relief of back symptoms was not as satisfactory as the relief of the radiating leg pain. Seventy-three per cent. of the patients in whom the spine was fused and 52 per cent. of the patients without fusion had no back symptoms. The rest had complaints of backache or weakness of varying severity. In a small series in which insurance compensation was involved, 45 per cent. of the patients have been returned to their original occupation.

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SPINE FUSION FOR PROTRUDING INTERVERTEBRAL DISCS

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During the six years since Mixter and Barr published their first work on protrusion of the intervertebral disc as a cause of low-back pain and sciatica, there has been a growing consciousness of this condition and a growing use of laminectomy in its treatment. The literature on the subject very often suggests that the clinical picture is typical, the diagnosis easy and definite with or without lipiodol or air myelography, and that the only treatment to be considered is laminectomy with the removal of the offending portion of the intervertebral disc.

Certainly if protrusions of the intervertebral disc are common today, due to tears of the annulus fibrosus or actual herniation of the nucleus pulposus, they must have existed to the same extent before the last six-year period. If these protrusions cause symptoms today, they must have caused the same symptoms in the past and driven patients to the medical profession.

Before the era of recognition of disc protrusion, these cases would have been, in the most part, indistinguishable from cases of low-back pain and sciatica attributed to disturbances at the lumbosacral or sacro-iliac joints. At the New York Orthopaedic Dispensary and Hospital the sacro-iliac joint has not been considered a common cause of this syndrome, and the condition has been explained in most cases on the basis of instability or mechanical inadequacy of the lumbosacral joint.

In the years from 1914 to 1936 inclusive, those patients who were operated upon for low-back pain and sciatica were treated in the main by lumbosacral fusion. Since the development of the Ober fasciotomy in 1934, this procedure has been used, with or without spine fusion, but to a less extent than fusion alone. In 1937 laminectomy combined with fusion, and occasionally laminectomy alone, was done in cases where there seemed to be sufficient evidence of intraspinal pathology.

To date there have been thirty-three laminectomies. In three of these no pathology was found. The other thirty included ten protrusions of the intervertebral disc, five cases of hypertrophy of the ligamentum flavum, one extradural scarring, two neurinomata, one varicosity along a nerve root, and eleven cases of adhesive arachnoiditis. Four cases of protruding disc and two cases of hypertrophy of the ligamentum flavum had arachnoid adhesions as well.

This variety of conditions, not specifically diagnosed before operation, is evidence of the difficulty in accurate clinical diagnosis of intraspinal lesions. In most of these conditions, spinal puncture, with manometric

and total-protein determinations, is of very doubtful differential value. One case, an inoperable neurinoma of the cauda equina, showed a total-protein content of 250 milligrams per 100 cubic centimeters above the level of the lesion, and xanthochromic fluid was obtained from the lesion itself. In the other cases there was no evidence of block, and the protein determination gave no indication of the type of pathology later found at operation.

TABLE I

RELATION OF TOTAL SPINAL-FLUID PROTEIN TO PATHOLOGY FOUND AT OPERATION

Pathology	*Cases*	Total Spinal-Fluid Protein (*Mg. per 100 c. c.*)
Disc protrusions without adhesions	10	58
All disc protrusions (4 with adhesions)	14	64
Adhesions alone	11	68
All with adhesions	17	68
All without disc protrusion or tumor	18	68
No pathology	3	61

Satisfactory myelography would do much to establish a definite diagnosis, but the authors have not found air myelography satisfactory, and they consider lipiodol too potentially harmful a substance to be employed.

There may very well be cases of true compression of the nerve roots by protruding intervertebral discs, and certainly sciatic pain is occasionally associated with new growths within the spinal canal, but the greatest number of the authors' laminectomies revealed arachnoid adhesions between the nerve roots, or a disc protrusion small enough to allow the nerve roots adequate passage. They found only one large herniation, 1 by 1.5 by 2.0 centimeters, apparently causing considerable nerve-root compression.

It has not been proved that a disc protrusion which merely angulates one or more nerve roots can cause symptoms in the absence of motion. Certainly the nerve roots are normally angulated about many structures in their normal course, and the spinal cord itself is often severely angulated at a tuberculous kyphos without the production of any nerve symptoms.

If the small disc protrusions cause nerve-root symptoms, it is much more reasonable to believe they do so by repeated sliding of the nerve over this protrusion with spine motion, than that they actually compress a nerve which is free to move away from the protrusion.

It is even more reasonable to believe that the small disc protrusion may often be an inconsequential element of a grossly unstable mechanical system. The stability of the lumbosacral joint depends not entirely on any one feature of its structure, but upon the composite of several elements and their relationship. The nature of the arch articulations, the angle of the superior surface of the sacrum, the presence or absence of impinging spinous processes, and of anomalies such as partial sacralization of the fifth lumbar vertebra, as well as degenerative changes, must all be

considered. A small disc protrusion, occurring in 15 per cent. of Beadle's series of spines, would seem to be merely another factor to be considered in relation to the others.

Radicular pain resulting from a fractured vertebra or arthritic lipping with pressure on the nerve roots has long been known to be amenable to immobilization by bed-rest, or spontaneous or surgical fusion.

Before the era of laminectomy for sciatic pain, most of the cases of protruding disc and arachnoid adhesions were undoubtedly fused. Either these patients were cured by fusion or must still have their pain, and present neurological signs. Those who had intraspinal pathology of any nature should be found largely among those patients who had neurological signs before fusion.

A study has been made of 175 cases treated by lumbosacral fusion for typical sciatic pain. Neurological examinations in this group were often omitted or inadequately done before the possibility of protruding discs was known. However, forty-six patients were found who had some or all of the abnormal neurological signs, now commonly attributed to protrusion of an intervertebral disc.

These forty-six patients include those with but few neurological signs, as well as those in which there was a fairly typical picture of nerve-root compression, as evidenced by unilateral calf atrophy, weakness of the anterior tibial and peroneal muscles, absence of the ankle jerk, and sensory loss in the lateral calf. Some have had even more marked changes, with complete foot-drop. It has been possible to see twenty-seven of these patients for this study and they have been compared with twenty-one of those who had had laminectomy fusions.

The results in the group with spine fusions were almost the same as the results in the group with laminectomy fusions.

TABLE II

RESULTS OF OPERATIVE TREATMENT FOR SCIATIC PAIN

Treatment	*Cases*	Average Relief of Pain (*Per Cent.*)	75–100 Per Cent. Relief of Pain (*Per Cent.*)
Fusion	27	85	83
Laminectomy-fusion	21	84	87

Five patients have had laminectomy following fusion. Of these, one had a protrusion of the intervertebral disc above the fusion, two had arachnoid adhesions, and one had extradural scarring. The fifth had laminectomy done in spite of a pseudarthrosis of the original spine fusion, because the neurological signs were not thought attributable to a pseudarthrosis alone. No intraspinal pathology was found in this case. In these five cases there was none with any appreciable improvement following laminectomy.

An examination of the reontgenograms of the thirty-three patients who have had laminectomy, revealed twenty-seven lumbosacral joints

classified as severely unstable; five as mildly unstable; and one as stable. This last patient had the large disc protrusion previously mentioned.

A comparison with a group of cases fused for sciatica, without any abnormal neurological signs, shows very little difference in the roentgenographic findings of the two groups; nor is there any outstanding roentgenographic difference between the laminectomy cases where a protruding disc was found and those where it was not found.

In summary, the diagnosis of protruding intervertebral disc is not one easily made. Clinical examination, spinal puncture, roentgenograms, and air myelograms have, in the authors' hands, been ineffectual in establishing an accurate diagnosis. Further, spine fusion alone has accomplished in many cases, which were clinically indistinguishable from protruding-disc cases, as good results as laminectomy and spine fusion with the removal of the disc protrusion. Theoretically, the majority, if not all, of the cases of protruding intervertebral disc can be relieved by fusion alone.

The authors submit that the need for laminectomy in treating protruding intervertebral disc has never been established. They present presumptive evidence that spine fusion alone is as effective as laminectomy and fusion, and suggest that fusion alone, a theoretically sound procedure, may be the treatment of choice.

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NOTE

The paper on Sacro-Iliac Conditions presented by Marius N. Smith-Petersen, M.D., as part of the Symposium has not been received for publication.—*The Editor.*

LOW BACKACHE AND SCIATIC PAIN ASSOCIATED WITH SPONDYLOLISTHESIS AND PROTRUDED INTERVERTEBRAL DISC: INCIDENCE, SIGNIFICANCE, AND TREATMENT

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The occurrence of sciatic pain associated with spondylolisthesis will become of increasing significance, will influence diagnosis and treatment, and will once more demonstrate the importance of cooperation among the roentgenologist, neurologist, and orthopaedic surgeon. Low backache is the most common symptom of spondylolisthesis, and it is equally, if not more, common among patients who have protrusion of an intervertebral disc. Sciatic pain, on the other hand, is common among those patients who have protrusion of an intervertebral disc, and occurs among only a small percentage of the patients suffering from spondylolisthesis. Although it is true that knowledge of the incidence of sciatic pain in the presence of spondylolisthesis is very limited, recent developments have shown the need for careful consideration of the possibility of a coexisting protrusion of an intervertebral disc causing the sciatic pain. At The Mayo Clinic the diagnosis of protrusion of an intervertebral disc, causing sciatic pain, has been made in patients for whom a diagnosis of spondylolisthesis with backache also had been made, and in whom the findings at operation substantiated such diagnosis.

To determine the incidence of low-back pain and sciatica among patients suffering from spondylolisthesis, histories have been reviewed of 745 patients for whom such a diagnosis was made from 1918 to 1939 inclusive. Eighty patients, or 10.7 per cent., were found who were so afflicted. A much larger percentage of patients suffering from spondylolisthesis had vague referred pain and paraesthesia of the buttocks, hips, and thighs that were aggravated by activity, such as hard labor, standing for long hours at a time, or injury. The majority of the eighty patients who had associated sciatic pain were referred to the Section on Neurology for further investigation, because the orthopaedic surgeon thought that, from the degree of the symptoms, possibly other factors were involved than those in his diagnosis. The truth of this suspicion became evident as knowledge regarding the etiology of spondylolisthesis and its complications became greater. Table I demonstrates that sciatic pain is more common among males than females—78.8 per cent. to 21.2 per cent.—and occurs most frequently during the years of active life. Seventy-seven and five-tenths per cent. of the instances of sciatic pain occurred among patients between the ages of twenty and fifty years. The average age of these patients was 41.1 years; the average age of the males was 40.2 years ranging from fourteen to seventy-three; the average age of the females was 44.2 years, ranging from twenty-one to eighty.

TABLE I

INCIDENCE BY SEX AND AGE OF SPONDYLOLISTHESIS WITH BACKACHE AND SCIATIC PAIN

Age by Decades, (*Years*)	Total Patients		Male		Female	
	Number	Per Cent.	Number	Per Cent.	Number	Per Cent.
0– 9	0	....	0	....	0	....
10–19	1	1.2	1	1.6	0	....
20–29	19	23.8	15	23.8	4	23.5
30–39	26	32.5	23	36.5	3	17.6
40–49	17	21.3	12	19.0	5	29.4
50–59	11	13.8	8	12.7	3	17.6
60–69	4	5.0	3	4.8	1	5.9
70–79	1	1.2	1	1.6	0	....
80–89	1	1.2	0	....	1	5.9
Total	80	100.0	63	100.0	17	100.0

Trauma is an important medicolegal question in the presence of sciatic pain. Frequently the causation of a disabling sciatic pain is difficult to ascertain, and the employee and employer look to the physician for advice in settling compensation claims. When spondylolisthesis complicates the clinical picture, the question is even more perplexing and taxes the diagnostic ability of the most expert physician. As a rule, patients who have low backache and sciatic pain associated with spondylolisthesis have had symptoms for years; some had backache at the time of onset, and later began to suffer from acute sciatica following a strain or injury; others had no definite single incidence of trauma, but were engaged in occupations that entailed strenuous exertion. In a previous publication[1], the author has shown that in about 10 per cent. of the cases in which a diagnosis of spondylolisthesis had been made, the patient did not complain of pain referable to the back, and that the diagnosis was made incidentally during the routine physical examination or during roentgenographic examination of other parts of the body. Because the usual backache and paraesthesia found in association with spondylolisthesis are as a rule relieved by rest and aggravated by work, these conditions have often led to the opinion that the patient was a malingerer. When the picture is confused by sciatic pain following strain or injury, intraspinal pressure must be considered.

The author believes that patients afflicted with spondylolisthesis are more likely to have protrusion of an intervertebral disc than are those who have a more stable spinal column. Trauma is a definite factor in

TABLE II

GRADE OF SPONDYLOLISTHESIS WITH BACKACHE AND SCIATIC PAIN IN RELATION TO HISTORY OF TRAUMA

Grade	Trauma		No Trauma		Total	
	Number	Per Cent.	Number	Per Cent.	Number	Per Cent.
1.....	28	65.1	21	56.8	49	61.2
2.....	13	30.2	11	29.7	24	30.0
3.....	2	4.7	3	8.1	5	6.3
4.....	0	....	2	5.4	2	2.5
Total.....	43	100.0	37	100.0	80	100.0

this condition. In this series of eighty patients, forty-three, or 53.8 per cent. had a history of trauma. Eleven of the seventeen classified as having suspected protrusion of a disc gave a history of trauma. These eleven constituted 25.6 per cent. of the forty-three who had a history of trauma, and 65 per cent. of those suspected of having a protruded disc. The remaining six patients constituted only 16.2 per cent. of the thirty-seven who gave no history of trauma. It is the author's opinion that fusion of the lumbosacral region is desirable in those cases of spondylolisthesis, with symptoms of protruded disc in which the surgeon is unable to demonstrate the disc at the time of the operation. This fixation of the spinal column will prevent movement and slipping, and is the most certain method of preventing additional symptoms of backache and sciatic pain.

TABLE III

INCIDENCE AND LOCATION OF SCIATIC PAIN IN RELATION TO GRADE OF SPONDYLOLISTHESIS

Spondylolisthesis, Grade	Total Patients		Location					
			Right		Left		Bilateral	
	No.	Per Cent.	No.	Per Cent.	No.	Per Cent.	No.	Per Cent.
1.....	49	61.2	23	58.9	17	68.0	9	56.2
2.....	24	30.0	12	30.8	7	28.0	5	31.2
3.....	5	6 3	3	7.7	1	4.0	1	6.3
4.....	2	2.5	1	2.6	0	....	1	6.3
Total....	80	100.0	39*	100.0	25†	100.0	16‡	100.0

* 48.8 per cent. of total of eighty cases.
† 31.2 per cent. of total of eighty cases.
‡ 20.0 per cent. of total of eighty cases.

TABLE IV

COMPARATIVE INCIDENCE OF DIAGNOSIS OF PROTRUDED INTERVERTEBRAL DISC IN EIGHTY OUT OF 745 SPONDYLOLISTHETIC PATIENTS WITH SCIATIC PAIN AND BACKACHE SEEN FROM 1918 TO 1937, INCLUSIVE, AND FROM 1938 TO 1939, INCLUSIVE

Years	Cases	Suspected Protruded Disc	
		Number	Per Cent.
1918 to 1937	55	2	3.6
1938 to 1939	25	15	60.0
Total	80	17*	21.3

* 2.3 per cent. of 745 spondylolisthetic patients. Six of the seventeen, or 0.8 per cent. of 745 cases with suspected protruded discs, were operated upon.

The most common spinal anomaly which this study revealed was separation of the neural arch which was noted in twenty-nine patients, or 36.3 per cent. Evidence of spondylitis or hypertrophic changes was noted in thirteen, or 16.3 per cent., and of sacro-iliac arthritis in three, or 3.8 per cent. of the roentgenograms of the spinal column.

The incidence and location of sciatic pain in comparison to the grade of spondylolisthesis are presented in Table III. It is interesting to note that pain in 91.2 per cent. of all cases, and 89.7 per cent. with sciatic pain on the right side, occurred in the presence of Grades 1 and 2 of spondylolisthesis, the lesser degrees of displacement. In the cases in which the condition was bilateral, 87.4 per cent. of instances of pain occurred in Grades 1 and 2. Marked displacements (as represented by Grades 3 and 4), are accompanied by fewer, not, as might be expected, more complaints, than are the lesser grades, indicating that there may be some factor other than spondylolisthesis involved in the production of the sciatic pain (Table III).

The significance of sciatic pain which is associated with spondylolisthesis was discussed at The Mayo Clinic when operations for protrusion of intervertebral discs were begun. Investigation of this latter condition soon made it evident that suspicions were justified. A thorough neurological investigation, including spinograms, is now a routine procedure for those patients with sciatic pain for whom the diagnosis of spondylolisthesis has been made. This is especially emphasized for those who have diminution or loss of the Achilles reflex, with pain, numbness, or paraesthesia. As a result of cooperation between the roentgenologist, neurologist, and orthopaedist, the incidence of protruded intervertebral discs diagnosed in association with spondylolisthesis with sciatic pain has increased enormously since 1937 (Table IV).

Figures 1*a*, 1*b*, 2*a*, and 2*b* demonstrate why forward slipping of the body of a vertebra alone will not cause impingement on the nerve roots.

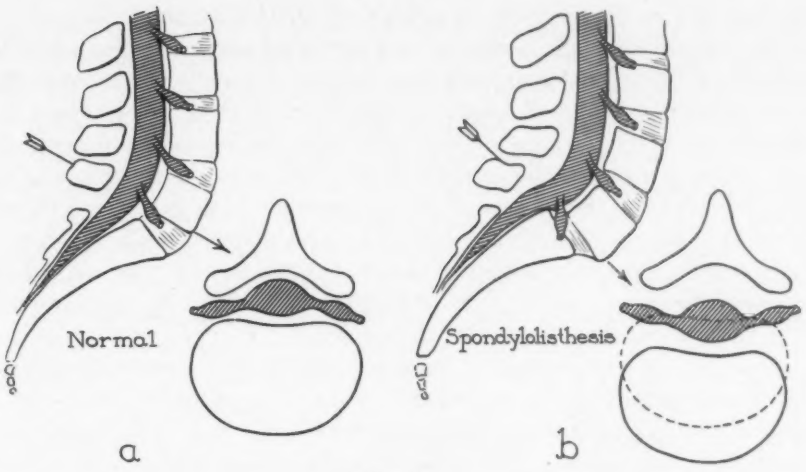

FIG. 1

Fig. 1*a*: Schematic drawing illustrating the normal relationship of the cauda equina and spinal column. On the right is a cross section of the spinal column in the lumbosacral region, showing ample space about the spinal cord.

Fig. 1*b*: The relationship of the cauda equina and spinal column in the presence of spondylolisthesis, grade two. On the right is a cross section of the spinal column in the lumbosacral region, showing the relationship of the spinal cord to the surrounding structures in the presence of spondylolisthesis, grade two.

The neural arch remains behind and, although the cauda equina and roots may be slightly stretched, there is enough room, in the large spinal canal, to prevent direct pressure on the cauda equina. In patients with the congenital types of spondylolisthesis, and in those in whom development was slow, there is rarely any paralysis, because nature compensates for stretch and pull. In the cases in which the injury is acute and severe,

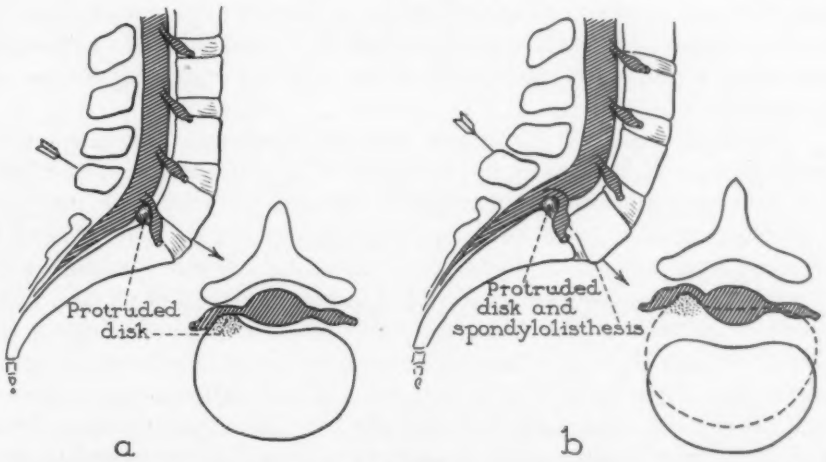

FIG. 2

Fig. 2*a*: Schematic drawing illustrating a protruded intervertebral disc without spondylolisthesis.

Fig. 2*b*: Protruded intervertebral disc in the presence of spondylolisthesis.

with fracture or rotation of a vertebra or vertebrae, it is obvious that evulsion of nerve roots, pressure of bone fragments, hematomata, and paralysis of the caudal segments may occur. Thus, it would seem that the common symptoms of spondylolisthesis, such as backache, pain, and paraesthesia referred to the buttocks and legs, are the results of instability of the lower portion of the spinal column with subsequent irritation and strain, rather than the results of direct pressure. The protruded disc, on the other hand, may cause direct pressure on the nerve roots. When the protrusion occurs in the center, bilateral distribution of pain may result, but when the protrusion is to the side, pain arises from the corresponding nerve-root distribution and sciatica results.

Conservative treatment of patients suffering from sciatic pain in the presence of spondylolisthesis, consists of rest by recumbency, use of external appliances and physical therapy measures, such as the application of heat and medication. Elevation of the flexed legs with traction so that the body is partially suspended, fixation in a plaster cast, or traction by means of double Buck's extension, and a firm bed, may aid in giving the patient comfort. Patients have been seen for whom all means of combating the backache and sciatic pain had failed, for whom conservative measures had been initiated without relief, and the author is, therefore, firmly convinced that such measures are of little permanent value. In spite of accurate diagnosis and explanation as to the cause of a patient's disability, there will always be those who refuse surgical treatment and who will have to be treated conservatively. The lumbosacral corset with reinforced steel stays, when worn persistently, and local applications of heat, are, when they are combined with medication, perhaps the most efficient non-surgical measures. In the present series of eighty patients, who had spondylolisthesis associated with a sciatic pain, conservative measures were carried out for forty-nine, or 61.3 per cent.; surgical fusion was performed for twenty-five, or 31.3 per cent.; and surgical fusion with removal of a protruded intervertebral disc was performed for six, or 7.5 per cent.

To insure relief of the sciatic pain and backache associated with spondylolisthesis, treatment must consist of immobilization of the lumbosacral region and relief of intraspinal pressure. At the Clinic, insertion of bone grafts alone in an attempt to relieve sciatic pain in such cases is not considered sufficient. Removal of the protruded disc combined with the added safeguard of lumbosacral fusion is the method of choice. The results from such a procedure have proved to be encouraging in the limited instances in which the patients have accepted this form of surgical treatment. There is no doubt that some of the patients may obtain a measure of relief from surgical fusion alone. The author believes, however, that such benefit would be partial only and never complete, and that the danger of recurrence of symptoms, the hazards of secondary operation, and the delay of prolonged disability would make it advisable to remove pressure from the nerve root in every patient with a proved

protrusion of an intervertebral disc. In the past this complication of spondylolisthesis was not recognized, and no doubt some patients who underwent the operation of spinal fusion, and received partial relief of symptoms, still have the offending discs that cause continued suffering.

A combined operation, the technique of which consists of laminectomy with removal of the protruded disc, and fusion of the lumbosacral portion of the spinal column, has given gratifying results. It is usually performed with the patient under spinal or general anaesthesia and requires two surgical teams. While the neurological surgeons remove the discs, the orthopaedic surgeons remove from the tibia the sections of bone for grafting. The time consumed in this first stage permits the immediate insertion of the grafts into the lumbosacral region and thus saves the patient an unusually long period of surgical treatment. In this procedure the neurosurgeon prepares the bed for the grafts and performs the laminectomy. Should there be considerable defect in the lamina, following the removal of the disc, the surgeon may use a graft of periosteum, sutured so that it covers the defect. To insure a strong bony support, two grafts are usually inserted, together with numerous fragments and cancellous bone that have been removed with a curet from the upper end of the tibia. The sides of the spinous processes, lamina, and facets are further roughened with a chisel or curet to insure bony approximation and ultimate fusion. The region which is usually fused consists of the third, fourth, and fifth lumbar, and the first two sacral vertebrae. The resultant mass of bone is the most secure fixation of the lumbosacral region that the author has been able to devise, and results from an application, with modifications, of the principles of fusion brought out by Albee and Hibbs. Roentgenograms taken subsequently show bony fixation. At the end of two weeks, during which time the patient has been kept recumbent and turned over three to four times a day, the incision is inspected and the stitches are removed. The patient is kept in bed for six to eight weeks following operation, and then is advised to wear a steel, reinforced, lumbosacral corset for six to eight months. A period of a year will elapse before the patient makes a maximal improvement, although there are some cases in which the patient is able to return to his usual occupation in much less than a year. When it is necessary for the patient to return home or to be moved some distance in less than six to eight weeks, a double plaster spica cast, applied so that it extends well up on the chest, will make this possible without much danger.

REPORT OF CASES

CASE 1. A man, aged forty-four years, reported to the Clinic on May 11, 1939, complaining of low backache, recurring shooting pain extending down the left leg, and foot-drop. About twenty-six years prior to his admission, he had often experienced, in athletic work, periodic pain in the back that sometimes shot down the left leg. This condition had continued to be aggravated by walking. He had been treated for arthritis. Five years prior to his admission an accident had aggravated the condition and two years later he had noticed progressive foot-drop developing. In spite of various treatments

and recumbency for a period of months, he was disabled and the pain had become progressively worse.

Examination revealed spondylolisthesis, grade one, of the third lumbar vertebra on the fourth, spondylitis with lipping, and left foot-drop; and absence of the Achilles and the external hamstring reflexes on the left, and of the Kernig and Lasègue signs. Examination of the spinal fluid, done elsewhere, had revealed a protein content of 71 milligrams per 100 cubic centimeters.

A left laminectomy, performed with the patient under spinal anaesthesia, revealed a large protruded intervertebral disc at the level of the fourth interspace, and marked hypertrophy of the ligamentum flavum. The protruded disc and the hypertrophied portion of ligamentum flavum were removed. The nerve root was found to be stretched over the disc and was swollen to about three times its normal size. A tibial graft and multiple bone chips were inserted to obtain fusion. The patient left the hospital on the fifty-first postoperative day, relieved of sciatic pain and wearing a lumbosacral support. He has had an uneventful convalescence, to the time of this writing.

CASE 2. In a previous publication, and in the exhibit presented before the American Academy of Orthopaedic Surgeons at the time of the meeting in Boston in January 1940, the author reported the case of a man, aged thirty-five, on whom he had operated for backache and left sciatica. A diagnosis of spondylolisthesis, grade one, of the fifth lumbar vertebra on the sacrum, and of protrusion of an intervertebral disc, was made. The protruded disc and hypertrophied portion of ligamentum flavum were removed, and a bone graft was inserted to produce fusion of the third, fourth, and fifth lumbar, and the first two sacral vertebrae. In this case the protruded disc had migrated from the fourth interspace to the lumbosacral region, and the fifth lumbar nerve had been stretched over it and become thickened.

CASE 3. A farmer, aged twenty-five, was admitted to the Clinic on June 19, 1939, complaining of backache with pain in the right leg. He stated that two years prior to his admission he had fallen twelve feet and landed on the buttocks. Following the accident he had experienced severe pain in the region of the coccyx, which disappeared in about two weeks. Four months prior to his admission, while engaged in heavy labor, the patient began to experience an aching pain in the lumbar region of the spinal column, which was aggravated by bending, lifting, weight-bearing, coughing, and sneezing, and which interfered with his sleep. Six weeks prior to his admission, sciatica developed on the right side, became severe in character, and extended into the heel. Furthermore, the patient began to lose weight rapidly, losing fifteen pounds (seven kilograms) altogether.

Examination revealed tenderness over the region of the fourth and fifth lumbar vertebrae, a positive Lasègue's sign, absence of the Achilles reflex on the right, and a total protein content in the cerebrospinal fluid of 35 milligrams per 100 cubic centimeters. There was an obvious depression of the lumbar portion of the spinal column, and some muscular spasm in the lumbar region with limitation of flexion. The roentgenograms revealed spondylolisthesis, grade one, with separation of the neural arch.

Following neurological and orthopaedic consultation, a diagnosis of spondylolisthesis with protrusion of intervertebral disc was made. The patient was advised to undergo laminectomy for removal of the disc, and bone graft for production of fusion of the lumbosacral region of the spinal column. He preferred to have conservative measures carried out first, and was sent home with a well-fitted lumbosacral corset.

The patient returned in about five weeks, and a right hemilaminectomy of the fifth lumbar vertebra was performed on August 3, 1939. A thickened ligamentum flavum and a huge classic protrusion of the lumbosacral disc were found. The latter was attached to a fibrous string of cartilage about 5 centimeters in length, thus relieving pressure on the nerve. Following the removal of the disc, a double bone graft was placed on either side of the spinous processes to produce fusion of the fourth and fifth lumbar and the first two sacral vertebrae. Multiple bone chips were packed around these grafts.

Convalescence was uneventful and the patient left the hospital on the forty-ninth postoperative day, wearing a lumbosacral support. The incision was healed, and roentgenograms showed the graft to be in good position. Seven months following the operation the patient apparently had made an excellent recovery.

CASE 4. A man, aged thirty-seven, was admitted to the Clinic on November 29, 1937, complaining of sciatic pain on the right side, of four months' duration, which had been aggravated by activity, and relieved by rest and flexion of the thighs on the trunk. Twenty-seven years prior to his admission the patient had fallen and injured the coccyx. Following the accident he had been treated for "rheumatism" of the left leg. This condition gradually disappeared. Since the onset of his symptoms, he had lost fifteen pounds (seven kilograms).

Examination revealed sciatic pain on the right side, and dental, tonsillar, and prostatic infection. The patient was advised to receive treatment for these conditions. Nine months later he again returned to the Clinic for examination because his pain had increased in severity in spite of the conservative measures that had been carried out. He still obtained relief following rest and flexion of the thighs on the trunk. The pain, which had been rather constant, was more marked on the right side, was aggravated by exertion, and noticeable across the right iliac crest and over the lateral aspect of the right calf. The lumbar portion of the spinal column was flattened, and there was muscular spasm, limited motion, and limitation of leg raising on the right. The protein content of the cerebrospinal fluid was 50 milligrams per 100 cubic centimeters. A diagnosis of protrusion of an intervertebral disc between the fourth and fifth lumbar vertebrae on the right, spondylolisthesis, grade two of the fifth lumbar vertebra on the sacrum, was made. Results of urinalysis and the reaction of the flocculation test were negative, and the blood count was within normal limits.

On December 6, 1937, excision of a small portion of the right lamina of the fourth and fifth lumbar vertebrae was carried out. The ligamentum flavum, which was hypertrophied between the two vertebrae was removed. The nerve had been displaced posteriorly by the protruded underlying disc. The nerve root was dissected out and the disc removed, which relieved pressure on the nerve. A bone graft was inserted to produce fusion of the fourth and fifth lumbar and the first two sacral vertebrae. Convalescence was uneventful. The patient was provided with a lumbosacral support.

CASE 5. A chauffeur, aged thirty-three, was admitted to the Clinic on November 7, 1938, complaining of recurring pain in the back and left leg of eleven months' duration. Instantly following a heavy blow in the left side of the lower part of the spinal column, he had experienced terrific pain, and on the day following, pain had occurred over the sacro-iliac region. This pain had lasted for several days, and later had extended down the left leg. It had been aggravated by coughing, sneezing, walking, standing, or lying down. He was most comfortable when sitting down. Results of roentgenograms, taken at that time, were reported to be negative. He had received physical therapy administered by a cultist. A week prior to his admission he had suffered from some form of infection, and had been told that he had a congenital defect in the spinal column.

Examination revealed a man of normal weight with normal blood pressure, pulse rate, and temperature. There was definite limitation of motion of the spinal column, with tenderness over the left sacro-iliac region, absence of the Achilles reflex on the left, and diminution of the knee and ankle jerks.

Following orthopaedic and neurological consultation, the patient was advised to undergo an operation, which was performed on November 16, 1938. A left hemilaminectomy revealed spina bifida with defect of the neural arch and a grade-one spondylolisthesis. The neurosurgeon noticed a posterior displacement of the first sacral vertebra, which was of a greater degree than that shown in the roentgenograms. There was a protruded disc at the fourth interspace, and both the fourth and fifth nerves were enlarged, as though they had been compressed. A bone graft was placed on the right of the spinous processes to produce fusion of the fourth and fifth lumbar and the first two

sacral vertebrae. The convalescence was uneventful and the patient left the hospital on the forty-ninth postoperative day. Subsequent roentgenograms revealed good position of the graft. About four months after the patient left the hospital he fell. This fall was followed by backache, limited to the region of the operation. No other symptoms occurred, and the pain disappeared in about two weeks. He had had no additional trouble with sciatic pain, and his general condition had improved. He was advised to continue to wear the lumbosacral support. He reported for observation May 23, 1939, and wished to return to work. He was advised to resume his usual occupation gradually and reported in June, 1939, that he had done so.

SUMMARY AND CONCLUSIONS

In the past two years the author has been impressed by the number of cases in which the patient had spondylolisthesis associated with sciatic pain and protrusion of an intervertebral disc. Protrusion of an intervertebral disc was diagnosed in fifteen of the twenty-five cases in which spondylolisthesis was associated with sciatica, and the diagnosis was confirmed in six cases in which operation was performed.

The treatment which offers the greatest benefit in the shortest period of disability and longest period of relief is surgical removal of the protruded disc and fusion of the last three lumbar vertebrae with the sacrum.

Cooperation of the roentgenologist, neurologist, and orthopaedic surgeon is essential in determining the exact situation of the lesion involved in the production of the backache, sciatic pain, and spondylolisthesis. Such cooperation has made it possible to diagnose the condition accurately and to give relief to the group of patients whose ailment heretofore has baffled the diagnostic efforts of even the most skilled surgeons.

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FASCIOTOMY FOR SCIATIC PAIN

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Fasciotomy for the relief of demonstrable fascial contracture of the thigh has its place in the treatment of chronic lame backs and sciatic pain. It should not be considered as a "cure-all", and is to be recommended only after a careful examination has been made and logical conclusions have been reached; otherwise, a helpful procedure will fall into disrepute. This paper contains a report of eighty-six patients from our clinic on whom fasciotomy has been done for the relief of sciatic pain. Practically all of these patients had chronic lame back and in several cases this was of several years' standing. The first patient was operated upon in May 1934, and the last one in January 1940. In thirty-one a bilateral fasciotomy was performed at one operation, in fifteen the bilateral fasciotomy was performed on the two sides at different times. There were forty unilateral fasciotomies,—twenty-seven left, and thirteen right.

In the bilateral fasciotomy group of thirty-one patients, sixteen are perfectly well, thirteen have shown slow, steady improvement, and one is moderately relieved. In the group of double fasciotomies performed at different times, seven patients are well, seven have shown marked improvement, and one is slightly improved. In twenty-seven patients with fasciotomy on the left, twelve have had excellent results, eleven are improved, one slightly improved, and two have received no help. Of the thirteen patients with operations on the right side, six are well, two improved, two slightly improved and three are not improved. In the eighty-six cases, forty-one have shown excellent results, thirty-three improvement, five only slight improvement, and five no improvement. There is no record in two cases.

The first patient had a fasciotomy on the right side in May 1934, and this was followed six months later by a fasciotomy on the left side for meralgia paraesthetica. The patient has remained perfectly well ever since. He had a severe lame back which was cured after the first fasciotomy.

Relief of the sciatica, following operation, may be obtained immediately or after a variable length of time up to a few months. The lame-back symptoms may continue for a longer period. The shortest history of sciatica was six weeks. This patient had such a very severe attack of sciatic pain that he was unable to feed himself. He received marked relief following the operation and was perfectly well in one month. Practically every case gave a history of lame back, and the duration of the lame back and sciatica, in a great many of these patients, covered many years, in some instances more than ten years. The sciatica was not always continuous, but came on or was aggravated when the lame-back condition returned, and usually lasted for months at a time.

TABLE I

RÉSUMÉ OF RESULTS OF FASCIOTOMY FOR PATIENTS WITH SCIATICA FROM MAY 1934 TO MARCH 1940

Treatment	No.	Results					Time Required to Relieve Back Symptoms		
		Excellent	Improved	Slightly Improved	No Improvement	No Record	Longest (*Years*)	Shortest (*Months*)	Majority (*Months*)
Double Fasciotomy at one operation.....	31	16	13	1	0	1	2	4	4–8
Double Fasciotomy at two operations.....	15	7	7	1	0	0	2	3	3
Single Fasciotomy, left	27	12	11	1	2	1	1	2	3–6
Single Fasciotomy, right	13	6	2	2	3	0	1	2	6
Total	86	41	33	5	5	2	2	2	3–8

Two of the group had had sacro-iliac fusion—one single and one double—without relief. Three had had lumbosacral fusions with only temporary relief, and two with no relief. Fasciotomy helped the patient who had had double sacro-iliac fusion. In the single case in which there was a history of sciatica for twelve years, the fasciotomy, which was bilateral and performed six years after the fusion, resulted in complete cure. The three cases of spinal fusion were cured or improved by fasciotomy.

The abduction test was positive in every one of these patients and almost always present on both sides. In several instances, where the test was positive on each side and a unilateral fasciotomy for sciatic pain had been performed without relief, a later fasciotomy on the opposite side gave complete relief. There were a small number of patients who had sciatica later on in the opposite side and these insisted on having the operation on the second side. One of these patients had had recurrent sciatica for ten years and operation stopped his pain within a month. Four years later he had sciatica in the opposite leg for two or three weeks and insisted on the same procedure for that side. There was complete relief within three weeks.

There have been only a few cases with recurrences and these have usually been slight and of temporary duration, and responded to conservative treatment. In one case a ruptured nucleus pulposus was removed and fusion was done. The sciatica returned again and fasciotomy was repeated with relief.

The operation will, if properly performed, relieve a large number of cases of sciatic pain. In some instances there is early relief of the lame-back condition when associated with sciatic pain. It has been found

that the relief of low-back pain, stiffness, and the associated poor posture is often slow. This is probably due either to the fact that the inflammatory condition is slow in clearing up, or that the physiological curves, having been distorted for years by the bad mechanics of abnormal fascial pulls on the pelvis and low spine, do not return to a normal posture quickly. In addition, the spinal curves, in returning to a normal posture, must exert pressure and tension on muscles, fasciae, and ligaments which have not been subjected to normal conditions for years.

A great many of the patients with lame backs improve so rapidly that no protective supports are necessary, but for those who do not recover rapidly, a support for the back is indicated and will hasten recovery.

Fasciotomy is being performed now quite extensively, and many surgeons have a better record of results than the writer. It should not be done indiscriminately for all sciatic pain, because there are often lesions of the back, of the sciatic nerve, or within the spinal canal which cause pain in the sciatic nerve. Each case of sciatic pain must be studied carefully. It is to be remembered that conservative measures will still relieve a large proportion of these patients and should be tried faithfully. On the other hand, if a patient is suffering great pain which appears likely to continue for some time, fasciotomy can be considered a conservative procedure. It will not add to the length of convalescence, can be advocated as a measure for relief, and should be done on that basis.

The operation is now performed as follows: An oblique incision, four to six inches long, is made from the lower edge of the anterior superior spine downward and backward, to a point just above the level of the greater trochanter and just posterior to it. The skin and subcutaneous fascia are separated by clean dissection above, below, and posteriorly, until a strip about two inches in width has been dissected well back over the anterior surface of the gluteus maximus muscle. The fascia is now divided from the anterior superior spine well back over this muscle. The fascia gaps at the incision, and the flaps are dissected off the muscles for about one inch on each side. All intermuscular septa are divided. If there is a positive Ely's sign, the fascia surrounding the sartorius muscle is also divided. All loose tags of fascial tissue must be removed. The length of postoperative time in bed depends on the severity of the back symptoms and the rapidity with which the sciatic pain clears up.

THE RELIEF OF LOW-BACK PAIN AND SCIATICA BY RELEASE OF FASCIA AND MUSCLE

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The introduction of a new method of therapy should be preceded by a scientific approach to the problem at hand, founded upon anatomical and pathological research, and followed, if practical, by experimentation. When stripping of fascia or fasciotomy was proposed by the author in 1934 such an approach had not been made, although the two cases reported then had been in the nature of experiments. These observations were recorded because at that time it was generally accepted that the origin of most cases of sciatic pain was at the lumbosacral or sacro-iliac articulations. Treatment was directed toward relief of strain at these joints, and, when conservative treatment failed, arthrodesis was the common practice. Controversy centered chiefly upon the differential diagnosis between lumbosacral and sacro-iliac lesions, and finally reached an impasse when surgeons fused all three articulations. Manipulative treatment persisted in making its reappearance from time to time, but generally with the idea of correcting a subluxation, for it was usually followed by fixation.

These concepts were so dominant that the title of the author's first paper on fasciotomy was expressed cautiously, "Thoughts on the Relief of Sciatic Pain", for it was akin to heresy to express a belief that an extra-articular superficial focus of irritation could manifest itself in the syndrome of sciatica. At about the same time Freiberg and Ober announced opinions at variance with the then current views, and Steindler shortly afterwards clarified the reflex phenomena of sciatica upon an anatomical basis,—a posterior syndrome. Procaine injections were a natural sequence as a diagnostic aid and a therapeutic measure. Clinical reports, together with the pathological studies by Gratz, point to a sustained interest in the musculo-aponeurotic and ligamentous origin of low-back pain. One need no longer be hesitant in accepting a lesion of these structures as a primary source of irritation.

In discussing the rôle of fascia and ligaments in relation to low-back pain upon the basis of relief following fasciotomy, the author is conscious of being in an awkward position of proposing an operation without sufficient background, and then trying to rationalize it. It is conceded that fasciotomy is on a weak pathological foundation, and it is difficult to be convincing by mere testimonials of relief when many patients with sciatic pain present comparatively few objective signs of disability. Remissions are common, and the frequency of low-back pain as a manifestation of neurosis is recognized. One must be cautious, therefore, in the interpretation of results of operation. Only those patients whom the author has personally studied are included in this paper.

Since posterior stripping has relieved carefully selected cases of low-back pain or sciatica, one must conclude that the source of irritation lay in a superficial focus in ligaments, fascia, muscles, aponeurosis, or at their periosteal attachments. It is difficult to conceive that the operation corrects sacro-iliac or lumbosacral strain, or relieves a direct irritation upon the components of the sciatic nerve. It then becomes necessary to assume that there is a so-called fibrositis, which would appear to be the weak point of this argument, because there is no convincing proof that fibrositis is a pathological entity. Clinical evidence, however, appears so convincing that it deserves a place as one of the three most common forms of rheumatic disease,—atrophic arthritis, hypertrophic arthritis, and fibrositis.

Space does not permit a complete review of the evidence upon which we accept the entity "fibrositis". Our knowledge of the pathology of fibrositis is practically confined to the studies of Stockman in 1920, and few if any have attempted to corroborate or advance his work. It is said that the term signifies an inflammation of the white, fibrous, supporting tissue, and that the inflammation is characterized at first by a low-grade serofibrinous exudate. Later there are fibrous and tendinous contractures or capsular thickenings.

Fibrositis is distinguished from arthritis by persistently negative evidence of intra-articular disease, no systemic manifestations, normal sedimentation rate, and frequent complete remissions. Slocumb tentatively classifies two types,—a primary and a secondary. The primary type is unaccompanied by, and is independent of, any other definite disease, and presumably is attributable to unidentified infections or toxaemia. The secondary type is secondary to arthritis, trauma, or influenza. The present status is well summarized by editorial comment in the *Fifth Rheumatism Review:* "Although the term 'fibrositis' may seem vague to some and the syndrome it implies incompletely understood, application of this term (or one more suitable) . . . seems in order."

Symptoms of fibrositis are muscle tenderness, stiffness, tenderness over insertions, and pain, particularly when the part is put on a stretch. Pain may be accompanied or replaced by a referred pain felt in the area of the skin corresponding to the nerve root which conducts the afferent impulse, and tenderness to deep pressure in the muscles in the same segmental area.

These incomplete and brief generalizations on an extra-articular origin of low-back pain, and the suggestion that a fibrositis is the basis of the irritation, may be helpful in the selection of cases for posterior fasciotomy. It is regrettable that there is no reliable single test or sign. The criteria for operation are as follows:

1. *Chronicity.* In general it must be assumed that the patient is not suffering from an acute fibrositis as these cases usually do well and recover by the conservative measures of protection, physiotherapy, and possibly procaine injections. Operation is reserved for the chronic case

in which persistent symptoms or tension is not relieved by conservative treatment. Hence, only comparatively few of this large group are suitable for operation.

2. *Localized Symptoms.* The focus of irritation must be localized at or immediately surrounding the posterior superior spine or immediately below the posterior third of the crest of the ilium. Local tenderness at periosteal attachments must be differentiated from diffuse muscular tenderness. In other words there must be localized tenderness at the musculo-aponeurotic or ligamentous insertions. There may be, however, pain and tenderness along the segmental distribution,—that is, sciatica.

3. *Tension Pain.* Generally there must be pain on stretching or active contraction against resistance. Any active or passive motion which exerts tension or stretch upon the affected parts is likely to be painful. Passive relaxation is not painful. This implies that forward bending, particularly while the patient is standing, is limited and painful. Lateral bending away from the affected side may cause pain by stretching, and toward the affected side by the tension of active contraction. Straight-leg raising causes local pain and is limited. Passive extension of the lumbar spine does not stretch the posterior structures and must be free and without pain.

4. *Procaine Test.* A positive response of temporary relief following a local and not massive procaine injection should be sought before proceeding with operation. It is not a necessary postulate, however, since it is difficult—at least for the author—to infiltrate the periosteum satisfactorily to block all painful sensations. There were some patients in this series who were not temporarily relieved by local injection of procaine, but who later obtained lasting relief by fasciotomy.

5. *Exclusion of Other Causes of Pain.* A careful study of the patient must be made to be assured, so far as possible, that symptoms are not caused by an intra-articular lesion or another factor recognized as causing similar complaints. A coexisting and asymptomatic hypertrophic arthritis of moderate degree, revealed only by the roentgenogram, is not a contra-indication to fasciotomy. Fibrositis may be secondary and only a part of the entire picture of arthritis, but still be the sole cause of symptoms.

The purpose of the operation is to release tension of ligaments, fascia, and muscles attached to the posterior superior spine and the posterior third of the crest of the ilium. This is done by subperiosteal stripping laterally, medially, and inferiorly, together with division of the sacrospinalis fascia when tenderness or pain is present there. The technique has been described fully in previous papers and will not be repeated here as it has not been modified.

End results also were reported by the author in 1939 before The American Academy of Orthopaedic Surgeons and published. At that time the results of twenty-one cases of posterior fasciotomy were reported with lasting relief in sixteen. Since that paper was prepared five more operations have been done, resulting in complete relief in four. This

makes a total of twenty-six operations with completely satisfactory results in twenty, or 77 per cent., and partial relief in three, or 11.5 per cent. The cause of failure in two of three cases is known. One had a congenital deformity of the sacrum, and the other had a considerable degree of hypertrophic arthritis in the sacro-iliac joint. It is now obvious that neither case was suitable for the operation.

SUMMARY

Fibrositis is a provisionally accepted entity causing low-back and sciatic pain which may be relieved by posterior fasciotomy in selected cases not yielding to conservative treatment.

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THE FASCIAL ELEMENTS IN ASSOCIATED LOW-BACK AND SCIATIC PAIN

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The explanation of sciatic pain by a physiological method is excessively difficult; perhaps, even, to the extent of proving to be impossible, unless the pain can be definitely associated with an organic lesion involving the nerve or its radicles. Man is the only vertebrate to whom the erect position is the normal and constant one. The anatomical arrangement of the sciatic trunk in relation to the muscles, and, therefore, to the fasciae, is quite different from that of any other vertebrate; and, even in man there is frequent variation to be found in this respect. There is no means of knowing whether any other vertebrate is subject to what is called "sciatic pain". It is known in man by his psychic response, only. Local tenderness is not only an inconstant, but also an unreliable guide to it, and the patient's response is the one means of recognizing whether such pain has been relieved. The only one who knows to a certainty whether he has pain and to what degree, is the patient himself. The surgeon is limited in his judgment by the finding of corroborative physical changes *in loco*, on the one hand, and his appraisal of the patient's emotional and psychic reactions, on the other hand. Furthermore, there are some who will acknowledge that in their own experience there have been numerous instances in which relief from sciatic pain has resulted from non-operative, so-called "conservative", methods in patients whose history and physical examination did not serve to differentiate them from others in whom it was finally deemed necessary to employ operative treatment. The indication for operation was the failure of the conservative method as it had been used. This result was not predictable or the sequence of events would not have been employed.

Additional experience and study with respect to the lesions of the intervertebral discs, as well as inflammatory conditions in fascia and joint capsule, have modified somewhat the author's attitude on this subject, as stated in 1937. The same thing can be said concerning the lesions at the sacrolumbar articulation. This is, however, not to be taken as a retraction of what was said in that paper, but rather as furnishing ground for greater clarity where the need was even then apparent. The conclusion drawn from the study made with Vinke and published in 1934, was that spasm or contracture of the piriformis muscle may be a potent factor in the production of sciatic pain. The suggestion to cut the muscle was made by Joseph A. Freiberg, and it was felt that this might constitute a useful procedure and, at the same time, furnish corroboration of the theory which was being postulated. The conclusion

* Deceased.

appears to be justified. However, the procedure was not offered as routine and is not now so offered. It has become plain as the result of personal observation, as well as the recorded observations of others, that both success and failure have attended the employment of the different surgical attacks upon muscle and fascia which have been put forward, namely:

1. The stripping of the attachments of the glutei, proposed by Heyman.
2. Novocain or procaine injection at the site of tenderness,—the method of Steindler.
3. The incision of the fascia lata, as proposed by Ober.
4. The incision of the piriformis muscle.

In communicating the account of the anatomical studies concerning the relationship of the piriformis muscle and the sciatic trunk, in 1934, there was no element of self-deception. It was not assumed that anything was definitely proved concerning the pathological physiology of sciatic pain. Similarly, in the absence of a great volume of clinical material and, considering the difficulty associated with the personal and psychic factors which are involved, an extremely conservative attitude has been taken by the author and his associates as to the appraisal of the results obtained by the operative procedures. Much the greater number of cases of sciatic pain which have been observed in three hospitals and which are considered to be unassociated with organic intraspinal lesions, have responded to non-operative treatment, consisting chiefly of traction, followed by mechanical support and physical therapy. Twelve patients who have had section of the piriformis muscle have been satisfactorily followed. In nearly all of them, the relief of pain was immediate. In two instances some pain remained for several days. In two of the twelve, relief of pain was not secured; they are considered failures, and should not have been operated upon. Among the earliest cases there were three patients in whom absence or very great weakness of the Achilles reflex on the operated side was noted. The author would feel justified, today, in suspecting that these symptoms represented organic intraspinal lesions, possibly from intervertebral-disc damage. However, it is interesting to note that in two of these three patients, sciatic pain was definitely disposed of. The third case is recorded as a failure. In the first of these three patients, operated upon five years ago, the physician reports that the Achilles reflex is still absent, and that sciatic pain has not recurred, although two attacks of fairly severe low-back pain have been experienced. This patient is reported now as the subject of a definite proliferative polyarthritis. Both of these cases, however inconclusively, raise the question whether the sciatic pain may here be explained as the merely irritative effect produced by an organic lesion.

Cases in which there is definite evidence of lesions of intraspinal or other organic structures, and direct involvement of the radicles or trunk

of the sciatic nerve, may be eliminated from the discussion as they belong in a different and special category. The cases with which we must be here concerned are chiefly those of sciatic pain of long standing.

Much more experience is required to justify a pronouncement which may be trusted to have an abiding value. As has already been said, successful results have been observed by the author in a limited number of cases treated by the methods of Heyman, Ober, or Steindler. These methods have been employed when it was thought that they were indicated, but in numbers too small to be worthy of statistical report. Like the explanation offered for the results of the piriformis operation, however, their explanation is one of rationalization rather than of proof. It would appear that the establishment of indications for the various procedures must rest largely upon such explanation, or upon a basis which is personal and empirical. Once surgical intervention seems called for, however, it will have to be acknowledged that in other operations an attack is being made upon structures much less directly connected with the sciatic trunk than is the piriformis muscle. Here we are at least concerned with a structure which is always in direct contact with the sciatic nerve, even though not always to the same extent or in precisely the same way. Much more study and experience would appear to be essential before the establishment of precise indications for surgical intervention is accomplished either with regard to time or the choice of method. It must be granted that the indications will have to be set forth on a purely objective basis, and therefore, upon one which bears no prejudicial and personal connection with one's rôle in the development of a method. To this the author is definitely committed.

The proposal to cut the piriformis muscle for the relief of sciatic pain was originally put forth with the idea that this might be welcome as a symptomatic release, in certain cases which had failed to yield to non-operative methods. While the possibility of disease primarily situated in the muscle itself was granted, the operation was done in the absence of definite evidence of its existence. Muscle contracture of continuing character is most often secondary to a lesion situated primarily elsewhere. When it is primary in muscle or fascia, it is conceived to be the result of inflammatory disease,—myofascitis, or as the British say, fibrositis. In no instance in which microscopic study of excised piriformis tissue was made, was there a report of local disease. In the absence of this it is concluded that the rôle of the muscle in producing sciatic pain is secondary. The same thing will have to be said in the case of operation on other muscular or fascial tissue. Unless there is tangible evidence of disease in the tissue itself or in the dominant nerve control, a merely symptomatic release is unsatisfactory. The primary lesion will have to be recognized and subjected to suitable treatment. Such recognition often makes an operation of any kind unnecessary. Here, for the time being, at least, the case must rest.

THE ARTICULAR FACETS IN RELATION TO LOW-BACK PAIN AND SCIATIC RADIATION

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INTRODUCTION

Numerous authors in the past thirty years have emphasized the possible importance of the articulations of the vertebral articular processes in low-back pain and in sciatic radiation. The publications of von Lackum, Danforth and Wilson, Ayers, Putti, Williams, Ghormley, and others between 1925 and 1933 showed renewed interest in the articular facets as possible causative factors in the production of sciatic pain. Indeed, Ghormley in 1933 coined a very expressive phrase, "the facet syndrome", which promised to stimulate further investigation of the rôle of the facets.

The recognition of the clinical application of the findings of Schmorl and Andrae concerning the protrusion of the nucleus pulposus, by Stookey, Mixter, Barr, Love, and many others, has temporarily at least turned the attention away from the facets and the plausible theories of their part in the production of sciatica, to the intensely interesting problem of the moment,—the protruded disc.

Recently, Ayers (in 1935) and Putti (in 1937) have again reported clinically and anatomically, respectively, the lumbosacral joint and its facets as factors in low-back pain. To the unprejudiced observer, well acquainted with derangements of the low back, posterior protrusion of the nucleus pulposus as the causative agent of low-back pain and sciatic radiation will always be regarded as a definite factor which occurs in a minority of patients with the sciatic syndrome. In the author's series of studied cases previously reported less than 20 per cent. show any neurological evidence of direct nerve irritation. The excellent response to conservative treatment in the cases without neurological signs, verifies our contention, previously expressed, that 80 per cent. of the cases of low-back pain with sciatic radiation are on the basis of referred pain, and not direct nerve irritation.

It is the purpose of this paper to present a review of the possible rôle of the vertebral articular facets in the production of low-back pain and sciatic radiation.

ANATOMY

It is important to recognize that the articulations formed by the vertebral articular processes (the articular facets) are true joints provided with a complete capsule which is lined with a definite synovial tissue. Closely associated with the mesial aspect of the capsule are the ligamenta flava. Piersol states that the outer portions of the ligamenta flava are

FIG. 1-A

Third and fourth lumbar vertebrae articulated to show asymmetrical facets on the right side. There is marked osteophytic proliferation on the superior surface of the lamina of the fourth lumbar and on the inferior surface of the facet, resulting in contact of the cephalic portion of the superior facet with the inferior surface of the lamina of the third lumbar vertebra.

FIG. 1-B

Detailed view of the superior facets of the fourth lumbar vertebra, showing more clearly these changes and the notch on the superior surface which engaged the lamina of the third lumbar vertebra.

inseparably attached to the loose articular capsules, preventing, by their tension, the occurrence of folds. They encroach on the sides of the capsules toward the canal, and *take the place of muscle* in preventing the capsule from being nipped between the articular surfaces during movement. The ligamenta flava are also valuable, according to Cunningham, because they restore the articular surfaces to their normal position with regard to each other after movements of the spinal column.

In general, the lumbar superior articular processes are stout, oval, curved plates of bone, fused in front with the roots of the laminae, with their concave articular surfaces vertical and turned medially. The inferior articular processes lie on either side of the root of the spinous process supported on the inferior margin of the laminae. Their articular surfaces, oval in outline, convex from side to side, and plane from above downward, are turned laterally. The inferior articular surfaces are closer together than the superior, so that, when articulated, the superior articular processes embrace the inferior articular processes of the higher vertebra. The inferior articular processes of the fifth lumbar are farther apart than the other lumbar vertebrae and their articular surfaces are directed more backwards than inwards and display less concavity. The superior articular surface of the first sacral has a vertical articular surface, of circular or oval form, which is concave from side to

FIG. 2-A

Anatomical specimen demonstrating a dissection of the posterior primary division of the third and fourth lumbar nerves. The median branch can be traced in close proximity to the inferior surface of the articular facet, winding around it and even giving periosteal branches to it, but with no observable connection between the nerve and the capsule. The nerve terminates in the sacrospinalis muscle and is primarily a motor nerve. The lateral branch is shown to the right with a small piece of muscle attached.

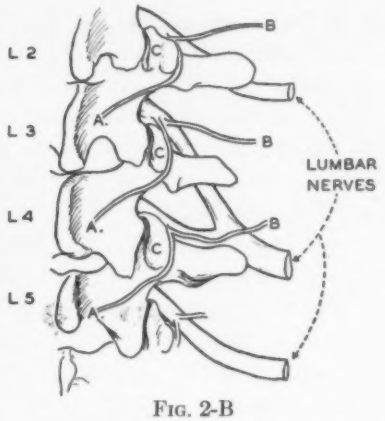

FIG. 2-B

Schematic drawing from the anatomical specimen.
A: Median branch of the posterior primary division.
B: Lateral branch.
C: Facet articulation.

side, with the general inclination backwards and a little medially.

Willis has pointed out that developmental variations of the articular processes in size, shape, and angle of projection, occur particularly in the lumbosacral region. He also states that poorly developed and asymmetrically formed articular processes strongly suggest mechanical instability and susceptibility to ligamentous injury.

Putti stressed the importance of asymmetrical development of the articular facets, regarding it as an anomaly of articular tropism.

Goldthwait recognized two well-known variations from the normal skeleton which are liable to produce backache,—(1) the tall slender visceroptotic type with long narrow flexible spine and flat type of lumbosacral facets, and (2) the short thick heavy type in whom the diarthrodial facets are large and crescentic and limit the motion of the vertebrae, particularly in flexion. Goldthwait pointed out that if the facets were asymmetrical, the movements were irregular.

Brailsford, in a review of 3000 roentgenographic studies of the lumbosacral spine, found that 57 per cent. of the lumbosacral facets pointed backward, 12 per cent. pointed inward, and 31 per cent. were mixed or asymmetrical.

More recently Putti, in a study of the anatomy of the vertebral apophyses, pointed out that there was a general shape of the articular por-

tion of the facets as described by anatomists, but that often the facets were flat and sometimes asymmetrical. He found that the inclination of the articular surfaces increased in the lumbar region from the first to the fifth with the minimal inclination usually 20 to 30 degrees in the upper lumbar area and 40 to 50 degrees in the fifth lumbar. Asymmetry with frontal facets on one side and sagittally placed facets on the opposite side occurred, particularly in the fifth lumbar, in six of seventy-five cases observed by Putti.

We have had the opportunity, through the kindness of Professor McCotter of the Department of Anatomy, and my associate Dr. Batts, to examine a large number of lumbosacral articulations. It has been expressed by Putti, and it is also our observation through roentgenograms, that there is a great frequency of facet abnormalities in the fourth lumbar. More frequently, distinct differences occur in the lumbosacral area and, phylogenetically, the frequency of abnormality should be greater in this region.

A careful measurement was made of 100 fifth lumbar inferior articular facets and first sacral superior articular facets. The variation in the sacral facets was reduplicated, within our ability to measure in degrees, exactly in the inferior fifth lumbar facets, so our findings are expressed only in the study of the superior articular facets of the sacrum.

FACET STUDIES

It is obvious that in general there is a normal for the inclination of the sacral facets, with an average angle of about 50 degrees from the sagittal plane. The average of inclination in this series agrees within a degree with Putti's findings. It will be seen by Table II that 79 per cent. of the cases did not vary 10 degrees from this inclination. Twenty-one per cent., however, showed as much as 11 to 30 degrees increase of inclination. One out of every five specimens showed an appreciable asymmetry.

FIG. 3

A fifth lumbar body demonstrating asymmetrical facets, osteophytic process at pedicle, and facet protruding into the spinal foramen just at the site where the fifth lumbar nerve winds around the body of the vertebra.

Facet Motion

It is stated in most anatomies that "flexion and extension are free in

TABLE I

ANGLES IN DEGREES OF SACRAL ARTICULAR FACETS WITH THE SAGITTAL PLANE IN ONE HUNDRED DRIED SPECIMENS OF SUPERIOR SACRAL FACETS

Angles	Average (*Degrees*)	Variation (*Degrees*)
Right	51.41	19– 87
Left	52.47	17– 90
Right and Left	103.88	55–177

the lumbar region, especially between the third, fourth and fifth vertebrae where the lumbar curve is the sharpest; lateral inclination is also very free between these same vertebrae. The shape and position of the articular processes of the lumbar vertebrae are such, that very little rotation occurs in this region." [8] Morris points out that there is always some space in which horizontal motion can occur around an axis down through the central part of the bodies and intervertebral discs, but it is very slight. Professor McCotter is convinced that this is an accepted, but somewhat

TABLE II

PERCENTAGE OF VARIATIONS OF NUMBERS OF DEGREES BETWEEN THE ANGLES MADE BY THE RIGHT AND LEFT SACRAL ARTICULAR FACETS WITH THE SAGITTAL PLANES

Variations (*Degrees*)	Cases (*Per Cent.*)	
0	9	79
1	9	
2	11	
3	6	
4	4	
5	8	
6	13	
7	5	
8	4	
9	9	
10	1	
11–15	15	21
16–20	2	
21–30	4	

erroneous impression. He feels that much more rotation occurs in the lumbar spine normally than has been thought possible. He bases this contention, first, on the shape of the facets, which are so constructed as to allow rotation, and, second, on the definite evidence in a number of skeletons of wearing of the articular processes and the bodies of the vertebrae, which could be explained in no other way than by rotation in the lumbar spine. (See Figures 1-A and 1-B.)

Von Lackum, in an anatomical study of thirty cadavera with eighteen grossly asymmetrical lumbosacral facets, states that when the articulations are asymmetrical they result in unequal rotation, a factor that also contributes to the weakness of the part. When there is associated with this condition a poor muscular or ligamentous development, or when there is a settling down of the superior articular facets onto the inferior facets from any cause, a very potent reason for disability or abnormality is established.

The Articular Portion of the Facets

Putti has made a comprehensive study of the size, shape, and inclination of the articular surfaces of the facets. There is as much variation in the true articular surface, as is found in the asymmetry of the entire facet. The area of the articular surface is usually 20 by 18 millimeters, but is commonly somewhat larger in the lumbosacral region. In normal conditions, the diameters vary from 8 to 10 millimeters in one direction, and from 9 to 11 in the other. Under this limit, Putti feels hypoplasia of the process exists. Not infrequently there is an asymmetrical development of the articular portion of the facets on the corresponding sides, with a difference in size as great as 6 to 8 millimeters in the transverse diameter.

The articular surfaces are covered with hyaline cartilage, usually of uniform thickness, but sometimes thinning toward the center. Putti also found evidence in the articulation of the facet of synovial villi which were extremely variable in shape, size, and appearance, frequently having two lobes, sometimes the size of the head of a large pin, sometimes leaf-shaped. They are rich in blood vessels.

The capsule of the articular facets is richly ennervated with sensory fibers, according to von Luschka. Most anatomists state that the primary posterior division of the spinal nerves supply the facet capsule, but that occasionally the recurrent nerve of the anterior primary division is the only sensory supply. At our request Professor McCotter and Dr. Strong dissected carefully the lower lumbar and lumbosacral nerves, and traced the medial branch of the posterior primary division of the third, fourth, and fifth lumbar nerves posteriorly. The individual nerves had a characteristic pathway, the medial branch curving in close continuity around the inferior margin of the facets, with some fibers extending into the periosteum of the facets, but continued as a motor nerve to supply the sacrospinalis group of muscles, without actual evidence of contact with the capsule of the facets. It is possible that the innervation of the capsule in this case came from the recurrent branch of the anterior primary division. Figures 2-A and 2-B demonstrate this dissection, showing the motor supply of the sacrospinalis from the individual lumbar nerves. The lateral branches were also dissected and also proved to be motor nerves.

The sensory nerve supply of the capsule of the facets is sufficiently well determined, to support the conception that irritation of the capsule of the lumbar articular facets could well produce pain stimuli which, through

FIG. 4

Thirteen dried specimens of the fifth lumbar vertebra are arranged to show the inferior articular facets. The variations in size, shape, and inclination of this unselected group are characteristic. Osteophytic proliferation about the vertebral border is demonstrated in a number.

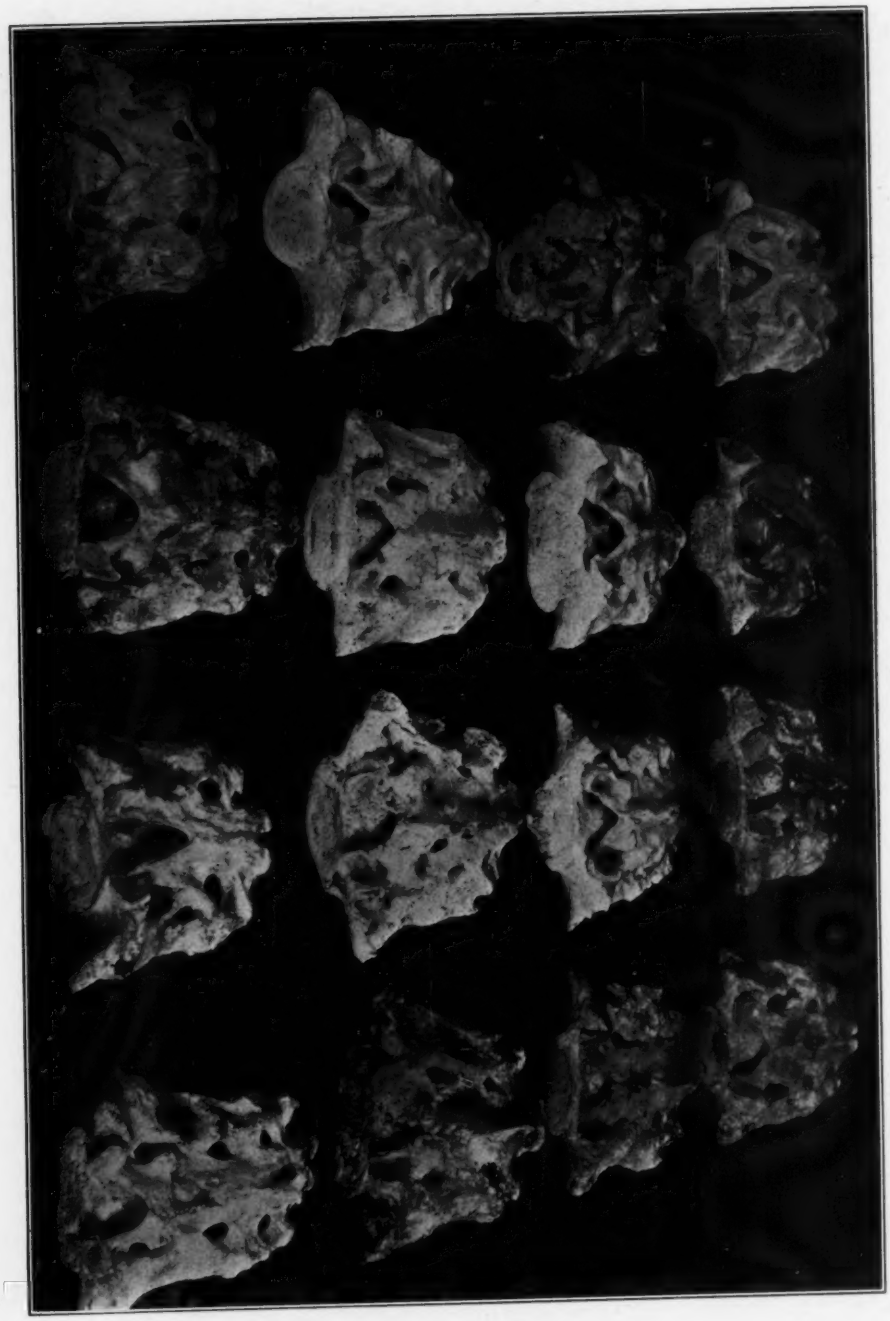

FIG. 5
Sixteen dried sacra illustrating the superior articular facets.

the primary posterior division, could return to the central nervous system and produce referred pain through the dermatomes of the involved nerves, which correspond exactly with the pathway of sciatic radiation,—namely, the fourth and fifth nerves. Thus sciatic symptoms on a referred mechanism, but along the same pathway as direct nerve irritation, can conceivably be produced. The author[4] has previously presented his theory of postaxial radiation as referred pain, not necessarily produced by direct nerve irritation.

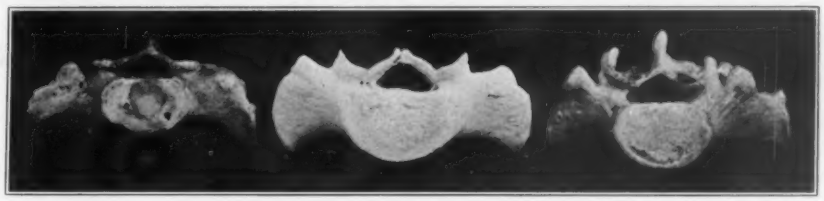

FIG. 6

Three specimens of the sacrum demonstrating the wide variation of inclination of the facets.

The anatomical relationship of the fifth lumbar nerve to the superior articular facet of the sacrum as the nerve passes through the intervertebral foramen has been stressed by various authors. The vulnerability of the intervertebral foramen by its alteration in size and shape through the mobility of the intervertebral disc and the articular facets has been excellently expressed by Danforth and Wilson. The increased encroachment of the intervertebral foramen in pathological conditions—such as narrowing of the lumbosacral disc, arthritis of the lumbar facets, and asymmetry of the facets with unilateral abnormal motion—has been described in most convincing manner by Ayers[3], Williams, and Ghormley. Anatomical support for the possible injury to the fifth lumbar nerve root in the intervertebral foramen seems as definite as the evidence of posterior protrusion of the nucleus pulposus and its relationship to the fifth lumbar nerve intraspinally, but pathological evidence is lacking to substantiate the anatomical.

The importance of the frequent clinical finding of narrowing or reduction of the fifth lumbar intervertebral disc, producing subluxation of the facets, in the necessary adaptation of the articular facets to this mechanical alteration of their function, has been well demonstrated.[10, 35] To allow for the approximation of the fifth lumbar body to the sacrum, the superior facets of the sacral vertebra must encroach toward the inferior vertebral notch of the fifth lumbar, even, in some instances, to the extent that they are stopped by contact with the lamina or the sacrum.

PATHOLOGICAL CHANGES

The anatomical evidence of the possibility of participation of the facets in the production of low-back pain and sciatic radiation has been

FIG. 7-A

Roentgenogram showing asymmetrical facet between the third and fourth lumbar vertebrae. On the left side the facet is much higher and smaller.

FIG. 7-B

Oblique view shows loss of clear view of facets of the fourth and fifth lumbar vertebrae on the left side.

greatly enhanced by further anatomical evidence of pathological changes and by roentgenographic and clinical findings which are sufficiently sound to warrant greater interest and investigation in the facet syndrome.

Sicard, quoted by Putti, stated that the intervertebral foramen is the cross road of neuralgia, and any condition which modifies the contents or the container (the intervertebral foramen, or the nerve or other structures within the canal) at once induces a painful reaction which is referred distally to the sciatic nerve. Putti for years maintained that sciatica is a neuralgia caused by a pathological condition of the intervertebral foramina and especially of the intervertebral articulations, the articular facets. It was his opinion that idiopathic sciatica is essentially the result of vertebral arthritis involving chiefly the articular facets.

Putti and Logròscino, in a recent anatomical study of seventy-five preparations of the lumbar and sacral regions, found that apophyseal arthritis is seen very frequently. These authors excepted individuals under thirty years of age who manifested no evidence of arthritis; they excluded six more, four of them in their early thirties, who had only minimal evidence of arthritis. In the remaining specimens, arthritic

FIG. 7-C

On right side the facets are clearly visible all the way to the first sacral vertebra.

FIG. 7-D

Lateral view shows normal appearance with normal disc spacings.

lesions in various phases of development were found constantly. Arthritis of the facets is rare under thirty years of age, but is progressively more frequent and severe with age. Intervertebral arthritis is more common between the third and fourth, and fourth and fifth lumbar vertebrae, and a little less common, surprisingly enough, in the lumbosacral region itself. Arthritis of the facets is more commonly associated with the asymmetrical facets. Lesions of the articular capsules were found in fifty-seven of the seventy-five cases, appearing usually in the upper and mesial part of the capsule with oedema, granular ossification, calcification, and adhesions between the capsule and the meningeal covering of the nerve root adjacent to it.

Changes in the synovial membrane were found analogous to changes in other joints in a degenerative process. Hypertrophic villi, of three to four times normal size, with chondrification and ossification occasionally in the villi; detached chondral bodies; and ulcerative areas, with complete loss of cartilage, were observed. Degenerative changes of the articular surfaces with thinning and fibrillation of the cartilage, and osteophytic marginal proliferation were frequently seen.

FIG. 8-A

Frontal view showing a saddle-shaped facet between the fourth and fifth lumbar vertebrae on the left side, and frontal type facet on the right side.

FIG. 8-B

Left oblique view further demonstrates the saddle-shaped facet.

Putti and Logròscino stated that they were particularly interested in the changes noted anatomically, which were not observable in roentgenograms. They presented, from their anatomical knowledge, certain signs noted in the roentgenogram which will enable the observer to recognize this form of vertebral arthritis roentgenographically, earlier than was previously possible.

Anatomical evidence is ample to demonstrate that the facets in the lumbosacral-joint area may and do suffer from all of the ills that are inherent to joint structures. Putti demonstrated changes in the synovial membrane, and free bodies in the joint that might conceivably produce symptoms akin to those of knee-joint derangement. The frequency of asymmetry of articulations required to function simultaneously must be a factor in repeated joint trauma. A mechanically unsound joint in the hip or elsewhere shows reaction to irritation as the patient ages, and so must the asymmetrical and mechanically poor facets. From Putti's studies he concluded that arthritis of the articular facets is the rule in practically all patients over forty years of age.

FIG. 8-C

Right oblique view shows normal facets.

FIG. 8-D

Lateral view shows normal disc spacing.

ACUTE TRAUMA TO THE FACETS

It seems necessary to discuss briefly the characteristic roentgenogram of fissure formation—usually of an inferior articular facet—found in a patient with back symptoms or accidentally in roentgenograms. This lesion has naturally led to the question of congenital or traumatic etiology. It is probable that the majority are the result of anomalous development. Fracture may occur, but usually is found only with evidence of fractures elsewhere in the spine. The analogy of this fissured process to the os trigonum and os naviculare is so striking that the author concludes that the etiology must be similar.

To evaluate properly the part the facets play in the production of low-back pain, postmortem studies of the lumbar and lumbosacral area of the spine are essential. Such studies may eventually be obtained in the larger medical centers, but are not yet available. Until such objective proof is obtained, we must rely on clinical and roentgenographic evidence and the response to treatment, and, therefore, the facet syndrome will remain a theory and not a fact.

The author is constantly confronted with problems of low-back pain,

often without sciatica, but with a history of sudden onset of pain, and sciatic scoliosis associated with marked muscle spasms, with no neurological findings, and frequently, no sciatic radiation. Many of these cases present changes which fit in so well with the anatomical factors mentioned in this paper, that it is most probable their symptomatology is based on the facet syndrome. A brief history of two cases is illustrative of this point.

CASE 1. A neurosurgeon, aged forty, had a second attack of acute, incapacitating, low-back pain, with a marked list. The leg signs were negative except an Ober's sign on the right side which disappeared as improvement set in. The neurological examination was entirely negative.

Roentgenograms showed a normal lumbosacral disc. The only abnormalities noted were: (1) The frontal view showed asymmetry between the third and fourth lumbar articulation with malformation of the left facet, and spina bifida occulta of the first and second sacral vertebrae. (2) The lateral oblique views denoted an obliteration of the facets between the fourth and fifth lumbar, and the fifth lumbar and first sacral vertebrae, which very probably demonstrated only asymmetry of the facets.

CASE 2. An electric welder, aged thirty-four, complained of chronic low-back pain since a fall of twenty-four feet, six and one-half years before, which injured both ankles. Lumbosacral pain was worse while lying down, relieved by motion, and aggravated at times while at heavy work. The clinical examination was essentially negative except for lumbosacral tenderness.

Roentgenogram of the frontal view showed a left asymmetrical facet between the fourth and fifth lumbar vertebrae and failure of complete closure of the lamina of the fifth. The oblique view showed the saddle-shaped facet on the left side.

The two cases are indicative of a great number of similar cases, where symptomatology may well be based on the facet syndrome. Their response to conservative treatment is excellent, but recurrence of difficulty is very possible. Fusion of the affected area usually results in complete relief.

CONCLUSIONS

1. The anatomical possibilities for the articular facets to play a more or less active part in the production of low-back pain with or without sciatic radiation are obvious, but pathological evidence is not yet sufficient to make this conclusion a fact.

2. Clinical evidence, in case of low-back pain with or without sciatic radiation, of pathology in or affecting the articular facets, requires serious consideration of the articulation in all cases of this syndrome.

3. The frequency of arthritis in the facets as age progresses, and the frequency of low-back disturbances in similar age periods, are coincidental facts of very probable significance.

4. Sciatica is a symptom complex, in which the syndrome is the only constant factor; the causative factors are variable and may be single or multiple, but the rôle of the facets should be considered in every case.

5. Rotation of the lumbar, and particularly of the lumbosacral articulation, occurs to a greater extent than anatomists have recognized. In this region it takes place chiefly in the facets, instead of in the intervertebral discs as it does in rotation in the thoracic region.

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